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TRANSACTIONS

OF THE

AMERICAN PEDIATRIC SOCIETY

TWELFTH SESSION

HELD AT WASHINGTON, D. C., MAY 1, 2, AND 3, 1900

EDITED BY

WALTER LESTER CARR, M.D.

VOLUME XII.

REPRINTED FROM

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CONTENTS

List of Presidents v
List of Officers for the Year 1900 v
List of Meeting Places v
List of Officers for the Year 1901 vi
List of Members for the Year 1901 vi
Minutes of the Twelfth Annual Meeting 1

I. Presidential Address. The Ambulatory and Hospital Management of the Gastrointestinal Derangements of Infancy in the Summer Months Among the Poor of Large Cities. By HENRY KOPLIK, M.D. 7

II. Clinical Observations upon the Operative Treatment of Tuberculous Peritonitis. By AUGUSTUS CAILLÉ, M.D. 17

III. Pancreatic Digestion of Casein. By B. K. RACHFORD, M.D. 29

IV. A Case of Rhachischisis. By T. M. ROTCH, M.D. 47

V. A Fatal Post-Otitic Cerebral Abscess with Amnesic Aphasia. By J. HENRY FRUITNIGHT, M.D. 56

VI. The Treatment of Hydrocephalus by Craniectomy. By EDWARD P. DAVIS, M.D. 63

VII. Intestinal Obstruction Through a Loop formed by Meckel's Diverticulum with Ligamentous Attachment. By IRVING M. SNOW, M.D. 67

VIII. Three Cases of Head-Nodding and Head-Rotation in Rachitic Infants. By D. J. MILTON MILLER, M.D. 72

IX. Nasopharyngeal Disease in Pediatric Practice: A Clinical Study. By FRANCIS HUBER, M.D. 81

X. Perforation of a Tuberculous Bronchial Lymph Node into the Trachea. Sudden Death. By A. CAILLÉ, M.D. ... 94

XI. Enteric Fever in Childhood. By A. D. BLACKADER, M.D. 98

XII. Exclusive Soup Diet and Rectal Irrigations in Typhoid Fever. By A. SEIBERT, M.D. 118

XIII. Two Cases of Fatal Lead Poisoning. By ALLEN BAINES, M.D. 122

XIV. General Subcutaneous Emphysema. By A. C. COTTON, M.D. 128

XV. Acute Nephritis Following Influenza. By ROWLAND GODFREY FREEMAN, M.D..... 132

XVI. Congenital Cardiac Malformation with Endocarditis and Anuria. By A. C. COTTON, M.D..... 142

XVII. Atresia of the Larynx Due to Traumatism, the Result of Faulty Intubation. By W. P. NORTHRUP, M.D..... 148

XVIII. Cough in Influenza Simulating Whooping-Cough (Pseudo Pertussis, Pertussoid). By F. FORCHHEIMER, M.D..... 152

XIX. Epidemic Paralysis in Children. By HENRY DWIGHT CHAPIN, M.D..... 158

XX. Malarial Coma in Children. By GEORGE N. ACKER, M.D.. 163

XXI. Poisoning by Vapo-Cresolene. By S. S. ADAMS, M.D.... 174

XXII. Hemorrhage into the Suprarenal Capsule in Still-born Children and Infants; Report of a Case Showing Rupture of the Sac and Escape of Blood into the Perirenal Tissues and Peritoneal Cavity. By S. McC. HAMILL, M.D. 176

XXIII. The Blood in Infancy and Childhood. By ALFRED STENGEL, M.D., and C. Y. WHITE, M.D..... 214

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1890. J. Lewis Smith, M.D.
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1892. Wm. Osler, M.D.
1893. A. D. Blackader, M.D.
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1895. F. Forchheimer, M.D.
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1898. L. Emmett Holt, M.D.
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1901. Wm. D. Booker, M.D

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MEETING PLACES

1888. Washington, (Organization), September 18.
1889. Washington and Baltimore, September 20 and 21.
1890. New York, June 3 and 4.
1891. Washington, September 22 and 25.
1892. Boston, May 2, 3, and 4.
1893. West Point, May 24, 25, and 26.
1894. Washington, May 29, and June 1.
1895. Virginia Hot Springs, May 27, 28, and 29.
1896. Montreal, May 25, 26, and 27.
1897. Washington, May 4, 5, and 6.
1898. Cincinnati, June 1, 2, and 3.
1899. Deer Park, June 27, 28, and 29.
1900. Washington, May 1, 2, and 3.
1901. Niagara Falls, May 27, 28, and 29.

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Buckingham, E. M., M.D.....346 Marlborough Street, Boston
Caillé, Augustus, M.D.....753 Madison Avenue, New York
Carr, Walter Lester, M.D.....68 West Fifty-first Street, New York
Chapin, Henry Dwight, M.D.....51 West Fifty-first Street, New York
Christopher, W. S., M.D.....408 Centre Street, Chicago
Churchill, F. S., M.D.....583 East Division Street, Chicago
Cotton, A. C., M.D.....1485 Jackson Boulevard, Chicago

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GRIFFITH, J. P. CROZER, M.D.....123 South Eighteenth Street, Philadelphia
HAMILL, S. McC., M.D.....1822 Spruce Street, Philadelphia
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Deceased

JOHN A. JEFFRIES, M.D.
Born, September 2, 1859,
Died, March 26, 1892.

THOMAS F. SHERMAN, M.D.,
Born, March 17, 1856,
Died, September 26, 1893.

JOHN M. KEATING, M.D.,
Born, April 30, 1852,
Died, November 17, 1893.

CHARLES WARRINGTON EARLE, M.D.
Born, 1845,
Died, November 19, 1893.

J. LEWIS SMITH, M.D.,
Born, October 15, 1827,
Died, June 9, 1897.

JOSEPH O'DWYER, M.D.,
Born, October 12, 1841,
Died, January 7, 1898.

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MINUTES OF THE TWELFTH ANNUAL MEETING OF THE AMERICAN PEDIATRIC SOCIETY.

Held at Washington, D. C., May 1, 2, and 3, 1900.

The meeting was called to order by the President, Dr. Henry Koplik, of New York. The minutes of the eleventh annual meeting were approved as published in the ARCHIVES OF PEDIATRICS.

The following members were present: G. N. Acker, M.D., Washington; S. S. Adams, M.D., Washington; A. D. Blackader, M D., Montreal; W. D. Booker, M.D., Baltimore; A. Caillé, M.D., New York; W. L. Carr, M.D., New York; H. D. Chapin, M.D., New York; W. S. Christopher, M.D., Chicago; F. S. Churchill, M.D., Chicago; A. C. Cotton, M.D., Chicago; F. M. Crandall, M.D., New York; E. P. Davis, M.D., Philadelphia; J. Dorning, M.D., New York; F. Forchheimer, M.D., New York; R. G. Freeman, M.D., New York; J. H. Fruitnight, M.D., New York; E. E. Graham, M.D., Philadelphia; J. P. C. Griffith, M.D., Philadelphia; L. E. Holt, M.D., New York; H. Jackson, M.D., Boston; A. Jacobi, M.D., New York; C. G. Jennings, M.D., Detroit; H. Koplik, M.D., New York; H. A. Lafleur, M.D., Montreal; C. F. Martin, M.D., Montreal: D. J. M. Miller, M.D., Philadelphia; W. P. Northrup, M.D., New York; C. P. Putnam, M.D., Boston; B. K. Rachford, M.D., Cincinnati; T. M. Rotch, M.D., Boston; I. M. Snow, M.D., Buffalo; L. Starr, M.D., Philadelphia; J. P. West, M.D., Bellaire; H. Williams, M. D., Boston; J. C. Wilson, M.D., Philadelphia; L. M. Yale, M.D., New York.

FIRST SESSION.—MAY 1.

The annual address of the President, entitled "The Ambulatory and Hospital Management of the Gastrointestinal Derangements of Infancy in the Summer Months Among the Poor of Large Cities," was read by Dr. Henry Koplik, of New York.

Dr. I. M. Snow, of Buffalo, reported a case of "Intestinal Obstruction through a Loop formed by Meckel's Diverticulum

with Ligamentous Attachment; Specimen," and showed a drawing of a similar case.

Discussion by Drs. Caillé, Adams, Christopher, Carr, Koplik, and Snow.

Dr. B. K. Rachford, of Cincinnati, read a paper on "The Pancreatic Digestion of Casein."

Discussion by Drs. Rotch, Caillé, Fruitnight, Cotton, Holt, Blackader, Koplik, Miller, and Rachford.

Dr. Augustus Caillé, of New York, read a paper entitled "Clinical Observations upon the Operative Treatment of Tuberculous Peritonitis."

Discussion by Drs. Fruitnight, Rotch, Cotton, Jackson, Koplik, and Caillé.

SECOND SESSION.—MAY 2.

Dr. Edward P. Davis, of Philadelphia, read a paper entitled "The Treatment of Hydrocephalus by Craniectomy."

Discussion by Drs. Rotch, Dorning, Koplik, and Davis.

Dr. R. G. Freeman, of New York, read a paper on "The Nephritis of Influenza in Children."

Discussion by Drs. Fruitnight, Dorning, Jennings, Rotch, Carr, Churchill, Caillé, and Freeman.

Dr. Augustus Caillé, of New York, presented a specimen and reported a case of "Perforation of a Tuberculous Lymph Node into the Trachea—Sudden Death."

Discussion by Drs. Rotch, Freeman, Dorning, Miller, Blackader, Fruitnight, West, and Caillé.

Dr. A. C. Cotton, of Chicago, showed a specimen and read a report of a case of "Congenital Cardiac Malformation with Endocarditis and Anuria."

Discussion by Drs. Adams, Blackader, and Cotton.

Dr. A. D. Blackader, of Montreal, read a paper entitled "Enteric Fever in Childhood."

Discussion by Drs. Cotton, Adams, Northrup, Griffith, Rotch, Wilson, Fruitnight, Graham, Freeman, Miller, Dorning, and Blackader.

THIRD SESSION.—MAY 3.

Dr. T. M. Rotch, of Boston, showed photographs and read a paper entitled "A Case of Rhachischisis."

Dr. J. H. Fruitnight, of New York, read the history of a case of "A Fatal Post-Otitic Cerebral Abscess with Amnesic Aphasia."

Discussion by Drs. Knapp, Chapin, Northrup, and Fruitnight.

Dr. W. S. Christopher, of Chicago, presented instruments and diagrams to illustrate a paper that he read on the "Measurements of Chicago School Children."

Discussion by Drs. MacDonald, Yale, Fruitnight, Cotton, Churchill, Chapin, Rachford, Griffith, Graham, and Christopher.

Dr. H. D. Chapin, of New York, read a paper entitled "Epidemic Paralysis in Children."

Discussion by Drs. Adams, Griffith, and Chapin.

Dr. W. P. Northrup, of New York, showed a specimen and read a report on "Atresia of the Larynx Due to Traumatism, the Result of Faulty Intubation."

Discussion by Drs. Miller, Cotton, and Northrup.

Dr. D. J. M. Miller, of Philadelphia, read a paper entitled "Three Cases of Head Nodding and Head Rotation in Rachitic Infants."

Discussion by Drs. Koplik and Miller.

Dr. S. S. Adams, read a paper on "Poisoning by Vapo-Cresolene."

The following papers were read by title:

Drs. A. Stengel and C. Y. White, of Philadelphia, "Studies on the Blood in Childhood."

Dr. G. N. Acker, of Washington, on "Malarial Coma."

Dr. F. Forchheimer, of Cincinnati, on "Cough in Influenza Simulating Whooping-cough (Pseudo Pertussis, Pertussoid)."

Dr. A. C. Cotton, of Chicago, on "General Subcutaneous Emphysema."

Dr. S. McC. Hamill, of Philadelphia, on "A Report of a Case of Ante-Natal Hemorrhage into the Suprarenal Capsule and Perirenal Tissue, Causing Death three days after Birth from Rupture of the Hemorrhagic Sac into the Peritoneal Cavity."

Dr. A. Seibert, of New York, on "Exclusive Soup Diet and Rectal Irrigation in Typhoid Fever."

Dr. A. Baines, of Toronto, on "Two Cases of Fatal Lead Poisoning in Children."

Dr. F. Huber, of New York, on "Naso-Pharyngeal Disease in Pediatric Practice."

EXECUTIVE SESSION.—MAY 3.

On nomination of the Council the following officers were elected for the ensuing year:

President, - - -	WM. D. BOOKER, M.D., Baltimore.
First Vice-President, -	FREDERICK A. PACKARD, M.D., Philadelphia.
Second Vice-President, -	J. LOVETT MORSE, M.D., Boston.
Secretary, - - -	SAMUEL S. ADAMS, M.D., Washington.
Treasurer, - - -	J. PARK WEST, M.D., Bellaire, Ohio.
Recorder and Editor, -	WALTER LESTER CARR, M.D., New York.
Member of Council, -	FLOYD M. CRANDALL, M.D., New York.

Minutes.

Place of Meeting—*Niagara Falls.*

Fee for Ensuing Year—*Five Dollars.*

The offer of the ARCHIVES OF PEDIATRICS to print the Transactions as in previous years, was accepted.

The Auditing Committee reported the books of the Treasurer correct.

Elected to membership, E. M. Saunders, M.D., St. Louis.

Resigned, B. Scharlau, M.D., and Wm. P. Watson, M.D.

Dropped, (according to the Constitution), Dillon Brown, M.D., and J. H. Musser, M.D.

Two applications were received, John Zahorsky, M.D., of St. Louis, and J. Madison Taylor, M.D., of Philadelphia.

On motion it was agreed that a resolution to assume an obligation of $25 for the expenses of the Paris Congress be laid on the table.

On motion of Dr. Christopher, it was unanimously voted to petition the Congress of the United States to provide adequate facilities for verifying weights, measures, and chemical apparatus.

WALTER LESTER CARR, M.D.,

Recorder.

THE AMBULATORY AND HOSPITAL MANAGEMENT OF THE GASTROINTESTINAL DERANGEMENTS OF INFANCY IN THE SUMMER MONTHS AMONG THE POOR OF LARGE CITIES.

PRESIDENTIAL ADDRESS.

BY HENRY KOPLIK, M.D.,

Attending Pediatrician to the Mount Sinai Hospital, New York.

Gentlemen of the American Pediatric Society:

I invite your sincere consideration to-day to a theme which is as broad and catholic in its bearing upon human happiness as any which can fall under the notice of the physician or layman. The care during the summer months of the vast number of infants and children of the poor in our large cities is a duty which in some countries the State and in our country private individuals assume. There is a distinct state and economic reason for this. It is not simply charity that impels us to care for these wards of humanity at large. It is our own happiness and the welfare of the State that is bound up with the welfare of the proletariat and its offspring. For this reason any fact, any suggestion which tends toward the improvement of present conditions with these little wards of ours among the poor, tends toward our own elevation, moral and material happiness. During the winter months we find among the crowded dwelling places of the poor in large cities those conditions which in an aggravated degree in the warm season cause a great mortality among the infant population. We find the housing together of vast numbers of human beings creates conditions of filth which, held in check by the lower temperature in winter, in a minor degree cause disease. In the summer, when temperature favors, these conditions act in a manner to cause frightful

mortality. In short, diseases caused by dirt infection are prevalent during the winter months but to a lesser degree than during the summer. We can explain this in great part by the fact that in winter the food, which is the principal source of infection to infants, is better preserved in spite of the constant factor of personal carelessness than in summer. In this country our statistics on this subject are very imperfect, but in France of all deaths below one year, one-half of the total mortality is due to gastroenteritis. In a small brochure by Madam Chaternikoff, we find that of twenty odd thousand infants below one year of life who died of intestinal trouble, between the years of 1891–98, in Paris, 3,639 were breast-fed and 18,818 bottle-fed infants. These figures are cited only to illustrate accurately conditions which exist to-day in every civilized land. In America and in my own city, New York, these conditions exist to a great degree, possibly greater than above indicated. The breast-fed infant is not exposed to any great or as many sources of infection as the artificially-fed infant. In the latter cow's milk is the substitute for the breast by common consent of all practical men. Yet before it reaches the infant it passes through so many channels, and is exposed to so many chances of infection, that we wonder the mortality is not larger than it really is. Milk, above all articles of diet, attracts to itself infection even if such infection is not directly introduced into the milk. The animals from whom the milk is obtained can first introduce infection. I do not refer to tuberculosis but to filth. The udder of the animal may be the means of introducing into the milk streptococci, which can cause virulent forms of diarrhea. Dairy dirt also may be introduced into the milk during milking. Again, in passing from utensil to utensil in commerce the milk is exposed to a thousand and one sources of infection. In the Congress of Pediatrists, held abroad in 1881, before the dawn of the proper understanding of the value of cleanliness in handling infant foods, great stress was laid upon cleanliness in the collection of the milk intended for consumption by the infant (Heubner). It remained for Soxhlet to show that the food must not only be brought to the infant clean and free from infection, but it must be preserved thus until consumed by the infant. By whatever means these things are attained to-day, all practical men will agree that the greatest light introduced into the dark and baffling problem of infant feeding remains absolute

cleanliness. As to the food itself, we find that the vast number of infants attain their majority through the most diverse methods of feeding. Each is lauded to the skies by the originator. I need only mention names such as Meigs, Biedert, Rotch, Escherich, Backhaus, Gaertner, Heubner, Soxhlet, Hoffman, to show that the successful feeding of the healthy infant can be carried out artificially in a variety of ways. The foundation of all artificial feeding must necessarily be the breast feeding; and yet in this natural method we find the greatest diversity in the quantity and quality of the food, tending to give equally brilliant results. We have a limited number of studies upon breast-fed infants which tend to show that as to quantities one infant will take only 89 caloric equivalents daily, another 126 caloric equivalents, and yet both infants be equally well nourished (Heubner). Thus, we cannot always fix upon absolute quantities. Again, it has been well established by Meigs and others that the proteids of the breast milk are constantly low, 1 to 1.5 per cent. The fat, however, varies largely in the same milk and in the milk of various breasts, and yet the children of each breast thrive. If we turn to cow's milk, we have a more difficult problem. We must not only dilute the milk, but there is a large waste even in thriving infants of the proteid constituents of the milk. This is manifested by an increase in the phosphorus in the excreta (Knöepfelmacher). Thus, it is not enough to simply construct the infant's milk on the breast proportions, but we must take into account the above waste and also the necessity of dilution.

As to dilution the leading minds differ. Budin, Chavaune, Comby, Drapier and at one time Escherich employed simply undiluted sterilized milk. Gauchas, Marfan, Heubner, Jacobi dilute the milk. Others, such as Meigs, Rotch, Escherich and Gaertner reconstruct the milk into proportions equal to that found in human milk. And yet in the vast number of infants outside of hospitals good results are attained by these clinicians, else they would not laud their methods. The truth is that with healthy infants mixtures of the most diverse nature will give results if the food is clean and the dilution carried to a necessary extent. Given, however, an infant suffering from ever so slight a gastrointestinal disturbance, it becomes a difficult problem to feed that infant. We have an *infection* added to the difficulties. It follows that in all our work we should sharply divide the well

from the sick infant, and the infant that is slightly ill from the severely ill infant.

That the gastrointestinal diseases, both mild and severe, are, in the vast number of cases, infections is the keynote of my theme. This is as true of the gastrointestinal disorders which occur to a mild degree during the winter months as of those that prevail to a startling and decimating extent during the summer season. We must no longer look upon the care of this large number of helpless sick as a problem of infant feeding simply. The time is ripe and the additions to our knowledge decisive enough to warrant the view that in treating the infants of our large cities during the summer months for gastrointestinal disorders, we are face to face with the problem of the treatment of infectious diseases just as much as we would be in the face of an epidemic of typhoid fever, measles, scarlet fever or diphtheria. Nay, our responsibilities are greater, for the mortality and suffering are much greater than in any of the above disorders. We teach the doctrine of isolation and disinfection and prophylaxis in the above infectious diseases, but it does not occur to the average physician to teach the ignorant mother that an infantile movement even of a normal character is infectious; that personal cleanliness on part of the mother in her maternal duties to her offspring is essential to its well-being and health. The food of the bottle-fed infant if contaminated by the hand of the mother is a frequent source of danger. How many physicians have ever warned the average mother to wash her hands after the baby's toilet is completed and before preparing the bottle for the baby's consumption? Few, I dare say. How many men who are daily thrown in contact with infants ill with diarrhea make a distinction even in their minds of the simple and very infectious forms of bowel complaint? Even in institutions here in America where infants are taken care of, but little attention is given to the scientific examination of the infants' diarrheal movement before treatment is inaugurated. And yet our progress has been so great in the simple clinical diagnosis of the bacteriological characteristics of the infantile movement, that to-day it is an aid to the proper and rational treatment of these diseases that such examinations be made. Prognostic value is very great if we know to-day that streptococci are absent from a diarrheal movement and treatment is not necessarily as laborious in the simple diarrhea of the coli variety as in

the severer forms of streptococcal infection. A distinction is in fact imperative to-day not only for the sake of our sick charges outside of our institutions but in institutions themselves to protect the healthy infants or those only mildly ill. This brings us to our present methods of caring for those infants in the summer months who are ill with diarrheal disease. We have two great methods of caring for these infants. The ambulatory method and the hospital or sanitarium method. By the ambulatoriums I refer to the great number of dispensaries and out-door services where these infants are treated. I think that the ambulatory treatment of summer diarrhea is thus far the most satisfactory. It does not presuppose conditions which I will show are conducive to increase rather than diminish the death rate. The sickest infants, those suffering from the severest forms of diarrhea, can be rescued by this mode of taking care of these infants. The large mass of sick is divided up among many institutions. The infants, to reach the dispensary, must be taken in the open. This tends to the good of the little patients who can be carried even in a febrile state out of doors without detriment. Let me describe the facilities of a large ambulatorium which for the past fifteen years has been under my own immediate supervision. When an infant suffering from diarrhea is brought to this institution it is undressed and examined carefully, the movements are studied grossly and examined microscopically and bacteriologically in a small laboratory in connection with the service. After such examination the little patient is then taken into a second room, which is well equipped with all apparatus for the treatment of gastrointestinal disorders. The treatment over, most careful directions are given as to diet for the succeeding twenty-four hours. The mother is told to bring the patient again. When the patient is convalescent to a degree as to permit the use of milk, it is given a proper food from the laboratory in connection with the service. This laboratory, now in its tenth year, feeds fully two hundred infants daily all the year round. The principles on which it is conducted are calculated to meet the greatest number of cases possible. The milk from which the food is constructed is as flawless as possible. Cleanliness is the all-pervading feature.

The effect of the food on the patient can thus be closely observed, and the food may be altered, or any other food substituted if the same disagrees with the patient. The mother is

encouraged to persist in treatment by weighing the infant from time to time in order to demonstrate the improvement. The most important element in the modern management of these dispensaries for the treatment of summer diarrhea is this department for infant feeding, which I think should be established in connection with every dispensary service. It is not enough to give the patient a prescription and directions to go and get a food at some station or depot, and then leave the little one to the tender mercies of the mother and the bottle of sterilized, pasteurized or any other food. This, it seems to me, is a very rapid way of getting rid of much work, but it does not aid if it does not harm the patient. The feeding laboratory should be on the spot or easily accessible to the clinic, and the physician can thus in person supervise the quantity and quality of the food, weigh the infant from time to time, noting progress. Healthy infants as well as sick infants are harmed by the unrestrained use, uncontrolled by the physician, of food, no matter how good. The prescription laboratory method in direct conjunction with a clinic on infantile diarrheal disease is the only true method of obtaining any definite results. Nor should any infant either in health or disease, obtain food at any station, except upon a physician's order. I have frequently found it impossible to successfully carry out the treatment of sick infants, because the mother would insist upon supplementing my milk quantities with milk obtained elsewhere. The infant thus was harmed by over-feeding. In my clinic we go as far as to furnish the water, in separate portions or bottles of six ounce capacity necessary for the preparation of simple albumen water in cases in which the infant is unable to take milk. In this way even an ignorant mother is impressed with the importance of attention to detail, even in such a simple substance as water. I mentioned the clinical laboratory; let me point out to those outside of this distinguished body, who may read my words, that the clinical bacteriological examination of the feces of sick infants taught to us by Booker, Escherich and their pupils is one of the greatest advances in the true understanding of the clinical aspects of these cases. It is not simply a fad, it is of great value, if the movement reveals the absence of bacteria of a known virulent character such as streptococci. It also aids us in returning to a milk diet if we see the gradual disappearance from the dejecta of these dangerous elements.

The dispensary service should be equipped with apparatus which will enable the physician to acquaint himself with the above practical facts. A great advantage in the ambulatorium, even of large size, is that there is rarely a crowding or heaping up of serious infectious cases such as I will show must necessarily exist in hospital services. The rapid treatment of a large number of serious infectious diarrheas in a hospital even if carried out with greatest care is sure to result in infections being carried from the serious to the mild cases, either through the hands of the nurses, assistants or instruments. In a dispensary it rarely happens that the serious cases are in great numbers. The mass of cases are mild, the serious cases can easily be separated and treated with care, leisure and cleanliness. Thus among hundreds of small dispensaries a great number of infectious diarrheas can be treated without danger of infecting each other and with much better results than in one large institution. The absence of hospitalism is also a great factor.

In the summer the sickest infant is much better off on the street, taken daily to the ambulatory clinic if the clinic is equipped as above, than lying in the most elaborate hospital ward alongside of other infectious diarrheas. The heaping up of infections is the great danger element. Septic diarrheas with pneumonia seem in my hands to have done very well with the ambulatory treatment. In a brochure by Paul Ignard, Paris, 1899, we see that the mortality of gastrointestinal disorders treated in the hospital wards of Paris was very high, the cases almost all of them died. The ambulatory dispensary cases, speaking more especially of atrophy with chronic gastrointestinal derangement, did much better. The tendency in the hospital ward is to develop septic complications, such as bronchopneumonia or of a simple diarrhea to rapidly develop streptococcic features.

One of the most interesting brochures in recent years bearing upon the great dangers of the crowding together of nurslings in wards of hospitals is that of Heubner, of Berlin (Sauglingsernährung und Sauglingsspitäler, 1897: Berlin). Those who fondly think that the problem of the treatment of these bottle-fed infants suffering from gastrointestinal derangement is solved by the food question alone, either in the sterilization or pasteurization of the milk, should read this very carefully elaborated monograph. Heubner shows that cleanliness in the

feeding, separation of nurses into classes, by which the nurse who performs the infant's toilet is not allowed to feed the baby, has reduced the previous mortality of 80 to 90 per cent. to 65 or 70 per cent. But it must be noted that here there is a halt. Why such a great mortality of 65 to 70 per cent., which in some months Heubner shows even mounted higher? It was the crowding together of infection and infectious cases. By isolation of very sick diarrheal cases, by the construction of barracks in which fewer infants were treated and more nurses attended to the infants, he hoped to reduce the mortality still more. And there we leave this painstaking clinician battling with a difficult and discouraging problem. Why treat these infants in hospitals or sanitariums at all? I think many will answer that there are a number of destitute infants who for some reason or other must be treated in this way. Their mothers, on account of the severity of the struggle for existence, cannot leave work and attend to their offspring at dispensaries. I think this problem of mortality will never be solved as long as the present hospital ward system exists. I have seen discouraging results in sanitaria in which wards accommodating only four or six infants existed. There was every improvement, every facility that money could obtain and science suggest, and yet the infants lay in their cribs and the charts showed a daily loss of weight. How will this problem eventually be solved? To my mind, and I have given the subject thought and study, it can never be solved except on the colony or cottage or camping system, very much in the same manner that pulmonary cases are treated. I can conceive of a colony of huts, with plenty of air and light, each holding two beds and in bad cases one bed, separated from each other by sufficient ground spacing arranged on a high tableland, not necessarily near the sea, but on the contrary inland, where the air is not harsh but free from the heat of the city streets. Such small, primitive huts can be taken apart, disinfected and put up anew with each season. They should be arranged over acres of land around a central administration building, in which the physicians and nurses live and in which the food is prepared. The laundry should be built apart from all else. The mothers should live in the huts and conduct themselves much as they would in their homes; take their infants out into the open if ever so ill. At fixed times they can come to the central station and obtain their food and that of

their infants, as at a dispensary. At a stated hour the doctor and his assistants will be at the main building, and once a day the infant can be locally treated and prescribed for and the mothers taught to give the food and medicine and also cleanliness in the toilet and handling of food. In this way the herding together of very sick infants is avoided and the ambulatory system is imitated as much as possible. Very sick infants, of whom there may be one or six a day, are to be especially looked after. If an infant is improving, it should not be held too long in such a colony, certainly not over a week or ten days. It is then sent home to be treated at the dispensary. In all this I have in mind the breaking up of the ward system and the substitution of the cottage or hut or camp system, and this of the most primitive character. I have made it a colony for sick infants. I think the system in some of our cities of taking two or three healthy children with a mother and one sick infant into sanitariums is wrong, and open to objection. I have seen the healthy infants in such places develop a dangerous diarrhea from a house infection, having come to the sanitarium in a healthy condition. As to the nursing, it would be more of a supervisory than of an active character. The mothers would be taught by competent nurses how to wash their hands after caring for the diapers and to give the bath, food, etc. In this way the mother is educated and is the nurse for her infant and feels she is doing something for it. She would not be introduced, as she is to-day, into a barrack-like room full of beds, with other mothers, with the depressent features of such a place. Her cabin or hut would have every comfort in a primitive way; a small stove for a wood fire in damp, cold weather. The drainage must be from high ground and well planned, so that among the adult population no typhoid fever or dysentery may arise. The vicinity of the seaside is not necessary, in my mind. I am not an enthusiast for the damp, bleak days at the seaside which cause little epidemics of bronchitis and pneumonia among the infants when, as the natives say, the air is "too strong." Inland, even if somewhat warmer, on high ground, with plenty of shade trees, seems to me to be preferred in cases of weak infants as a site for such a camp. In such a camp I also presuppose that the physicians in their equipment have a clinical laboratory and the feeding will be conducted on principles laid down in the ambulatory treatment. The old method,

in some places the present way, by which a dozen or more infants are placed in a ward, even if the infants be practically out of doors in a piazza enclosure, must certainly have been very discouraging to those of us who have had occasion to attend such a service. The infants do not seem to thrive. They seem to infect each other either through the nurses or assistants. They lose in weight and baffle all our efforts at progress.

An advantage of the camp system is that, should any infant develop measles, scarlet fever, or diphtheria, it is by the system itself at the time in a condition of isolation and need not be moved, nor need others be disturbed or the business of the camp interrupted. And now let me say, that all the above is in the sense of suggestion. I am anxious to have the opportunity to see this system tried. It has the advantage of simplicity; it inculcates the principles of isolation of cases from each other, not by ward space alone but by sunlight and air. It allows of the application of our most advanced ideas to individual cases and is not as expensive as elaborate hospital pavilions, which do not aid us but rather increase our difficulties.

I will not take up the care of healthy infants in this address except to say that personally I am not in favor of housing healthy infants in large numbers in-doors any more than sick infants. Diarrheas are sure to crop up no matter how great the care observed. Thus, it does not aid us if, in order to treat a sick infant, we must take into an institution the mother and one or two healthy young infants who may develop diarrhea of a dangerous character in the institution. We may improve the sick, but we endanger to an extent the healthy.

There are many more minor methods of caring for the sick and even healthy infants in our large cities during the summer months. I have only written of those lines of work into which fate has thrown me. I have endeavored to awaken an interest in new channels:—To inculcate the importance of gross differentiation of forms of diarrhea, of isolation of these various forms of cleanliness, of the dangers of overcrowding, and the evils of bringing the sick and healthy under one roof.

And here let me close, hoping that the future will open up a new scientific era in the management of the summer disorders of infancy among the poor which will contrast favorably with the discouraging history of the past.

CLINICAL OBSERVATIONS UPON THE OPERATIVE TREATMENT OF TUBERCULOUS PERITONITIS.

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By reason of its peculiar clinical behavior, tuberculous peritonitis claims in a high degree the interest of the physician and surgeon inasmuch as the brief exposure to the air or sunlight of a portion of the accessible infected area is apparently the starting point of a reparative or healing process in cases that have resisted other therapeutic efforts.

The opening of the abdomen is, as a rule, followed by an arrest of local disease symptoms, and may be followed by a disappearance of the tuberculous deposits on the peritoneum, as shown by certain cases in which the abdomen has been opened for some reason or other for the second time.*

Two points of special interest present themselves to the clinician:

1. Behavior of tuberculous peritonitis before the opening of the abdomen, or, in other words, the diagnostic features of the disease.

2. Its clinical behavior after laparotomy.

This report embraces not all the cases observed by me, but only such as were admitted to my service at the Babies' Wards, Post-Graduate Hospital, and carefully studied before and after operation and which had subsequently been under my observation for a period of from one to three years.

For the sake of brevity and to avoid repetition I will state that every case underwent a careful examination, including the examination of urine, blood, feces and puncture fluids;

* "Tubercular Peritonitis Cured by Laparotomy." Dr. F. Jameson, *Buffalo Med. Journal*, May, 1899.

and no special mention will be made in the brief histories whenever the co-existing conditions were found to be normal. Much of the laboratory work was done in the laboratory of the New York Post-Graduate School, by Dr. H. T. Brooks.

CASE I.—Maurice J., six years old; admitted February 9, 1897; sick for the past five months; gradual swelling of the abdomen, loss of flesh, no pain.

Status on Admission.—Weight, forty-one pounds; temperature, 100° with subsequent rise to 103°; pulse, 100; respiration, normal.

Physical examination showed normal conditions except as follows: The abdomen was swollen and somewhat tense, not painful on percussion or on palpation, and contained fluid. On auscultation much peristaltic unrest was noticeable. Double inguinal hernia also existed. A diagnosis of tuberculous peritonitis was made by exclusion, and between February 9th and April 16th creosote, creosote carbonate, guaiacol, ichthyol, and arsenic were exhibited internally and externally and by means of rectal irrigations, but without apparent benefit. On the contrary, a hard mass appeared soon after admission, in the scrotum, close to the testicles on either side, which slowly spread and was looked upon as a tuberculosis of hernial sac.

On May 16th the operation for double inguinal hernia was performed by Dr. B. F. Curtis, by the Bassini method. The peritoneum forming the hernial sacs on both sides and in the abdomen as far as the finger could reach when introduced into the inguinal rings, presented the usual pathological appearance of tuberculous peritonitis. The membrane was unusually thickened, covered with nodules and deposits of fibrin, and congested. There was some clear serum in the abdominal cavity; the testicles were healthy. The hernia was not congenital on either side. The wound healed in due time. I saw patient two years after operation and found him to all appearances perfectly well. Physical examination revealed no abnormality. It is to be noted that in this case the general abdominal cavity was not encroached upon at the time of operation.

CASE II.—Franz B., two and a half years old; admitted April 19, 1898; weight, twenty-five pounds; temperature, 99° with occasional elevation; pulse, 112; respiration, 36. Patient eats well and looks well.

A careful clinical examination was made, which was negative except as follows:

Abdomen symmetrically distended, no fluctuation. A firm tumor within the abdomen, on each side, and a third tumor in median line higher up; each tumor has well defined, sharp margins; operated upon April 27, 1898, by Dr. B. F. Curtis; no

previous medication. The tumors were found to be large tuberculous deposits in the omentum, firm and vascular. Intestines studded with tubercles of all sizes. No fluid in abdominal cavity. Tumors not adherent to any organ and no enlargement of mesenteric nodes. One independent tuberculous nodule was found high up in the omentum. Ten grammes of a 10 per cent. glycerin-iodoform emulsion was put into the abdomen and the same was closed. Convalescence was uneventful. Eighteen months after operation the tuberculous tumors can still be felt but are very much smaller. There is no other evidence of tuberculous disease.

CASE III.—Isidor B., two and a half years old; admitted February 28, 1897; on admission looked anemic but not jaundiced; said to have large stomach for one year; weight, twenty-six pounds; temperature, 102.4°; temperature curve shows irregular low fever; pulse, 140; respiration, 48.

Complete clinical examination negative excepting abdomen, which was enormously distended; two quarts sero-sanguineous fluid were removed by trocar. The child has lost weight and strength and has pain. Constipation is noticed but no vomiting.

Operation July 19th, by Dr. Coley. Showed multiple encysted pus cavities and detritus and characteristic miliary tubercles; the wound was drained; a subsequent counter opening was made by Dr. Lloyd. After a lingering illness the child died. The operation was not followed by any improvement.

CASE IV.—Declon B., four years, admitted August 8, 1898. Abdomen began to swell six months before admission, otherwise feels well. Clinical examination negative, excepting albumin and bloody epithelial casts in urine. Abdomen twenty-four and a half inches in circumference, distended with fluid; area of liver dulness extends from nipple to one inch below ribs. Weight, twenty-three pounds; temperature, 98° (subsequent irregular low fever); pulse, 124; respiration, 38.

Operation November 5, 1898, by Dr. Lloyd. Showed a characteristic tuberculous peritonitis. The abdomen was flushed with warm normal salt solution and closed; uneventful recovery. This boy is in very good health at the present time.

CASE V.—Elizabeth N., five and a half years old, admitted March 31, 1898. Her abdomen became large and tense a few months before admission to Babies' Wards; no pain. Weight, thirty-eight pounds; temperature, 69° (irregular low fever); pulse, 120; respiration, 24.

Careful clinical examination negative, except as follows: Encysted fluid in abdomen extending no higher than umbilicus; line of percussion flatness not changed by putting patient in Trendelenburg or other positions; dark brown fluid removed by puncture; gonococci in vaginal discharge.

Operation April 27, 1898, by Dr. B. F. Curtis. Peritoneum thick and adherent, large amount of dark brown serous fluid evacuated; adhesions broken by finger; intestines covered with miliary tubercles; abdominal cavity irrigated with saline solution and dried out; 10 per cent. emulsion of iodoform and glycerin used; abdominal cavity closed. The fluid evacuated had its source in a large cavity, which was separated from the general cavity above by adhesions which were not ruptured. A sinus from the abdominal wound persisted for about one year and closed spontaneously the middle of April, 1869. Directly after closure a cough set in, and on examination May 11, 1899, moist râles were heard over entire right lung. The sinus opened again and discharged; it was subsequently curetted by Dr. Wilson, and has closed completely. At the present the patient is well and has gained in weight.

CASE VI.—Julia D., three years old, admitted April 24, 1899. Twenty and a half pounds' weight on admission; temperature, 98° (fever curve irregular, from normal to 103° F.).

For two months before admission she complained of pain in abdomen, vomiting and constipation. The clinical examination was negative excepting abdomen, which was tender on palpation. Under ether the intestinal convolutions could be felt, also bands of tissue which proved to be adhesions.

The abdomen was opened April 24th by Dr. Dunham and a tuberculous peritonitis with intestinal adhesions found. The wound healed, leaving a fecal fistula with adhesions. Up to the present time her general condition is splendid. The fistula persists and will require further operative interference.

CASE VII.—Joseph F., six and a half years old; admitted June 15th. The boy's mother died of pulmonary tuberculosis. For six months past he has had spasmodic pain about the umbilicus. He is constipated, pale and has no appetite. A careful examination reveals nothing of note, excepting a tender abdomen. Weight, twenty-three pounds; temperature, 100.4° (irregular low fever curve); pulse, 100; respiration, 24.

The appendix can be felt, but is not as tender as other parts of abdomen. The percussion sound is that found over collapsed intestine.

Operation was performed July 5th, by Dr. Wilson, and an adhesive miliary tuberculosis of peritoneum was found.

The abdominal cavity was flushed with saline solution and closed. The boy is quite well at present writing.

CASE VIII.—Joseph C., two and a half years old, admitted May 17, 1898. Three weeks before admission the abdomen began to enlarge, and child was feverish. The scrotum began to fill when child cried; the hernia now remains down continually; skin muddy-looking. Weight, twenty-three pounds; temperature, 101.5° (irregular fever curve); pulse, 40; respiration, 36.

Abdomen distended, flatness on percussion, free fluid in peritoneal cavity; reducible right inguinal hernia; hemoglobin, 60 per cent.; red corpuscles, 5,417,000; white corpuscles, 6,250; urine, trace of albumin, hyaline casts. Operation May 27, 1898, Dr. B. F. Curtis.

Finger detected a large cyst cavity, extending to umbilicus, made up of small cysts containing considerable fluid; fluid evacuated; 10 per cent. emulsion of glycerin-iodoform introduced. The thickened peritoneum was closed with continuous catgut suture, skin also. A nodule from omentum proved to be tuberculous on microscopic examination; hernia not operated upon.

Examined May 11, 1899. Musical rhonchi on deep inspiration over scapula on both sides. Child pale but improved. Thickening of tissues in line of wound and line of old sinus. At present writing patient is pale and anemic, otherwise in apparent health.

CASE IX.—Isabella C., nine years; admitted January 24, 1898; weight, twenty-four pounds; temperature, 97° to 103° (irregular fever curve); pulse, 90-120; respiration, 30.

For past six months has had severe pain in abdomen and four to five stools during day and as many at night, losing flesh rapidly: Abdominal tenderness marked. Abdomen flabby; palpation reveals nothing noteworthy. No ameba or tubercle bacilli in stools. No plasmodia in blood. Red cells, 5,025,000. A careful examination showed no other abnormality. As the parents of the girl refused to permit an operation for the suspected tuberculous condition, all known methods of internal, percutaneous and rectal medication were persisted in for the greater part of two years.

In November, 1898, the girl looked very anemic and the abdomen was tender and tense, but her weight was thirty-six pounds. In March, 1899, her weight was down to twenty-eight pounds. She had much diarrhea and vomiting and intense paroxysmal pain, also some cough and general anasarca. In February, 1899, the parents of the patient gave permission to open the abdomen (Dr. Wilson). The intestines were found to be matted together by tuberculous tissue and the adhesions were found to be too tense to be broken up. There was no pus. The accessible portion of the abdominal cavity was flushed with saline solution and the abdomen was closed. The girl died in October, 1899, from exhaustion. No autopsy.

In this case pain and paroxysms, diarrhea and low irregular fever were the symptoms for the greater part of two years. Palpation of the abdomen revealed nothing abnormal.

CASE X.—Girl of nine, Flora B., who was afflicted with a not very extensive tuberculous infection of right lung apex and whose abdominal symptoms came on gradually. She had pain

in paroxysms, pain on pressure. Various dull areas next to tympanic spots in abdomen. Occasional fever and loose bowels. On abdominal section the intestines were found matted together, the interspaces were filled with a dark yellow fluid and some detritus. Microscope revealed tubercle bacilli. Iodoform-glycerin introduced; drainage; marked improvement, but died from general tuberculosis two years later.

The following three cases have also been under observation for at least one year, but I have since lost track of them and am unable to report as to their present condition, and whether they are alive or dead.

CASE XI.—Harry V., five years old, admitted October 12, 1897. Patient was apparently well up to two months before his admission to the Babies' Wards, when he lost his appetite and began to vomit; he coughed and expectorated; had fever and sweating and dyspnea. Weight, forty pounds; temperature, 100° (irregular fever curve); pulse, 140; respiration, 40.

A careful clinical examination revealed: abdomen distended, superficial veins prominent, fluid in abdomen; area of fluid dulness changes with position. Sonorous râles and rhonchi are heard all over the chest. He looks anemic and poorly nourished. Abdominal section by Dr. Curtis showed tuberculous peritonitis. Iodoform and glycerin introduced; closure of abdomen; perfect healing of wound; discharged improved, and lost sight of a year ago.

CASE XII.—Hattie V., four and a half years old, admitted March 2, 1898. Weight, twenty-eight pounds; temperature, 100° (irregular fever curve); pulse, 132; respiration, 32.

This patient had a tuberculous look or habitus, and it was stated that her abdomen had begun to swell some time before admission to the hospital. She was constipated, had fever, but no cough, and her abdomen was found distended with fluid and gas. Albuminuria was also noticed.

Operation by Dr. Curtis March 17, 1898. The peritoneum was found thickened, the bowels agglutinated, and much fluid was evacuated by breaking down adhesions. Miliary tubercles were seen in great numbers on loops of intestine and tuberculous elements were detected by Dr. Brooks, Post-Graduate Laboratory.

Iodoform-glycerin was put into abdomen and the latter sutured. Healing by primary union. General condition much improved after operation. Discharged in good condition, and lost sight of a year ago.

CASE XIII.—Frank O., three years old, admitted August 23, 1898. Weight, twenty-three pounds; temperature, 100.2° (irregular fever); pulse, 120; respiration, 28.

The child was sick, three months before admission into the hospital, with cough, distended abdomen, and alternating constipation and diarrhea. Bilateral bronchopneumonia; abdomen painful on palpation—contains fluid.

Operation by Dr. Lloyd. Discharged (unimproved); operation wound healed; general tuberculous infection; case lost sight of.

Résumé.—The diagnosis of tuberculous peritonitis is based upon the abdominal symptoms, such as distension, pain and disturbed bowel action, presence of fluid and loss of weight, and is made by exclusion, except in those cases in which the tubercle bacilli are found and then the diagnosis is positive. A febrile rise of temperature of an irregular type was found in all cases under careful observation. There is nothing characteristic about the temperature curve.

Cases of chronic non-tuberculous serous peritonitis present usually the features of an ordinary ascites, the abdominal fluid being free, whereas it is usually not free in the tuberculous variety. It is rare to find the tubercle bacilli by microscopic examination of puncture fluid. In doubtful cases the opening of the abdomen is indicated and will do no harm. Paroxysmal pain in the abdomen in children, in the absence of chronic appendicitis or abdominal fluid, is not indicative of tubercular disease and is frequently overcome by dieting and attention to and irrigation of bowels. (Worms, intestinal indigestion, membranous enteritis, etc.)

The tuberculin test was employed in Cases I and V; in the former with positive and in the latter case with negative result. I am unwilling to make a routine test with tuberculin in human beings in the present unsatisfactory state of our knowledge of its action.

To the three varieties of tuberculous peritonitis hitherto formulated by various observers:

1. Chronic tuberculous ascites (miliary form);
2. Fibro-caseous tuberculous peritonitis;
3. Fibro-adhesive tuberculous peritonitis,

must be classed a fourth variety.

4. Tuberculous peritoneal tumors.

Two such tumor cases have been seen by me. Israel* reports one and there may be others on record which have not come to my notice.

* "Geschwulst artige Tuberculose des Colon ascendens." *Deutsche med Woch.* No. 1. 1896.

The infection of the peritoneum can come about by way of the circulation or from the gastroenteric or genitourinary tract. Only one of the children here reported upon had a tuberculous parent (mother), Case VII, and it is not too far-fetched to assume that milk and meat of tuberculous animals are a frequent source of infection.

The cases here reported show the futility of medicinal treatment (most cases having been so treated before operation), and where some form of medication is followed by improvement or cure, one must not forget that spontaneous cures have also been reported and observed in cases presenting all the clinical evidences of the disease. Tuberculin and serum preparations were not exhibited as therapeutic agents.

Finally, the indication is early operation, which is no doubt of very great benefit to the patient when the tuberculous process is limited to the peritoneum. As regards the establishment of a complete cure, I am somewhat sceptical, because of the persistence of mild abdominal symptoms, of irritative catarrh or inflammation in bronchi, lungs and pleuræ, and intestines, in a number of cases which remained under my observation two years after operation. If at the time of operation we have co-existing tuberculosis of the lung or pleura, the ultimate results are unsatisfactory, although some improvement usually takes place for the time being.

DISCUSSION.

DR. FRUITNIGHT.—I would like to report a case, not in the person of a child, but still it is germane to the question both because it is unusual and interesting. The patient, a young woman of twenty, now under my care, presented herself with an ulcer on her hand, which was found on examination to be tuberculous, and on further examination it was found also that there was evidence of tuberculosis in the lungs, and furthermore there was also present tuberculous peritonitis. The ulcer on the hand was noticed, she says, in the shape of a little, hard swelling or lump. Unfortunately, the physician who first saw her, poulticed it and brought on suppuration and then incised it, and so possibly spread the infection through the system. The proper thing, it seems to me, would have been to have excised it, and then the general infection might have been prevented. The patient, when she consulted me, said she had had a more severe peritonitis, but it had improved under treatment. She

had been ill a number of months and had all the symptoms of tuberculous autoinfection. I agree with the reader of the paper as to the great value of early operation, having seen good results in the Children's Hospital of St. John's Guild, New York, but I do not believe the recovery will be permanent.

DR. ROTCH.—The subject of tuberculosis in infancy and early childhood is one of the most important we have to deal with, next to infant feeding. It has become more and more important every year. Out of some two hundred autopsies, where young children and infants had died from diphtheria, tuberculosis in some part of the body was found in seventeen or eighteen cases. The question of tuberculosis is exceedingly important to us who are paying especial attention to pediatrics.

In tuberculous peritonitis, what has seemed to me especially important is first the diagnosis between primary peritonitis in the sense that it originates in the abdomen, and tuberculous peritonitis that is secondary to tuberculosis elsewhere. For instance, a large number of cases of tuberculosis of the lung is associated with tuberculosis elsewhere, especially in the abdominal organs. The treatment of tuberculous peritonitis that is secondary to disease of the lungs is almost useless. When the tuberculosis of the peritoneum is primary, laparotomy is without question the proper treatment of the disease. We have had quite a wide experience with tubercular peritonitis, and the treatment is invariably laparotomy. We have never had any bad results from laparotomy. A very large number of cases, according to the class of cases we are dealing with, are not only benefited but are cured. I am possibly a little more optimistic than the last speaker as to the curative action of laparotomy. We do not know why, but we do know that we open the abdomen in a certain class of cases and they get well. I have followed the cases for years afterward and am sure that they recovered absolutely. That is true especially in one case where a boy who had tubercular peritonitis had been fed very largely the milk from a cow that on autopsy was found to be tubercular. The boy was cured by laparotomy and is perfectly well to-day. This brings up the question of what cases to operate upon. When there is primary tuberculosis of the peritoneum, we should operate; when the peritonitis is secondary the result is not so good. The cases that have been most benefited, in my experience, are those with effusion. They are the cases that almost surely get well. Secondarily we should operate where there is no effusion and there are simply tubercular masses or tumors, such as the reader of the paper has described. Laparotomy, it seems to me, is safer almost than giving castor oil. I say that only in a general way, because so much damage is done by laxatives. Surgeons should not hesitate to perform laparotomy. Then a large num-

ber of cases with effusion, which have been diagnosticated as non-tubercular, have really been tubercular, and the difficulty has been in not finding the tubercle bacillus. I have often in earlier days seen cases operated on where they did not find the tubercle bacilli, and so declared that the case was not tubercular. But I believe these cases were tubercular, because as the technique of finding the tubercle became more perfected, we have found them more frequently. We should never hesitate to operate in cases with obscure symptoms in the abdomen. Where the effusion has gone on the tubercular masses have broken down and the abdomen is full of pus, I have seen absolute recovery by laparotomy and washing out the abdomen. In those cases the bacilli were found. As we are finding tuberculosis to be so much more frequeut than formerly, the surgeons should be encouraged to operate. In the other cases, in which there is not effusion, the patients are benefited by laparotomy, and also they get well often without laparotomy. Where laparotomy has been refused I have seen the child get well when I was pretty certain of the diagnosis or had made the diagnosis as near as possible without exploration. When we have a small tumor in the abdomen I do not think we have a right to take it for granted that it is not tuberculous. I was very much struck with that in the case of a child of a surgeon in Boston, who is especially skilful in detecting tumors by palpation. He thought he detected an enlarged mesenteric node, and I unhesitatingly told him to have a laparotomy performed because we do not know how soon the mesenteric node when infected may cause further infection. As exploratory laparotomy is essentially harmless, it seems to me it should be performed in these cases. In this case a large tuberculous mass was found, tubercle bacilli were present, and a tumor was removed. That child's life, I believe, was saved, for in all probability it would have had secondary infection. What we want to do is to operate before secondary infection takes place. Another form of tuberculous peritonitis is where it is a secondary infection. These cases are, *per se*, cases of tubercular disease. As to the use of tuberculin, I have considerable faith in its use for diagnosis. The number of men opposed to the use of tuberculin would lead me to consider it as yet *sub judice* in treatment. But for diagnosis I would use it. What you need in the tests with tuberculin is a low range of temperature. When the temperature is 102° or 103° the result is not so good with tuberculin in diagnosis. When the temperature is low the diagnostic use of tuberculin is valuable and it does no harm.

DR. COTTON.—I fully agree with Dr. Rotch in the importance of tuberculosis as perhaps paramount to that of almost any other disorder in infancy and childhood. I am very glad I heard Dr. Caillé's paper, because I realize the extreme difficulty of making

a diagnosis in cases of suspected tubercular peritonitis, without tubercles, without nodules, possibly with effusion. I would like to submit a question to the Society. If I understood Dr. Caillé, he based his diagnosis sometimes upon an examination of the ascitic fluid. In my own experience that has been negative, even under control experiments. I have now two cases under observation. One, in which I strongly suspect tubercular peritonitis, has been shown a number of times in my clinic. But, as is often the case, in the absence of pain and tenderness, which is rarely the rule in my experience in these cases, there are no positive diagnostic symptoms. I am driven to the bacteriological findings for a diagnosis, but they often prove to be negative, and yet there is the ascites, and there is the tumor, and there is the tendency to emaciation. Now, the question I would like to ask is: How far we are justified in exploratory laparotomy for diagnostic purposes in these cases. I would like to be reinforced in my inclination, to insist upon exploratory laparotomy for the purpose of clearing up the diagnosis.

DR. ROTCH.—I do not see why we should not make an exploratory laparotomy in all cases. I would perform it and snip off a little piece of the suspicious tissue, and not examine only the fluid.

DR. JACKSON.—I would like to draw attention to one point in the diagnosis in these cases, and that is the absence of leucocytosis. Of course that will not separate the cases from typhoid fever, but it is an important point in making a diagnosis from ordinary peritonitis.

THE PRESIDENT.—In mentioning these tumors, due to tuberculosis of the peritoneum and enlarged mesenteric lymph nodes, a very important and large variety of tumor cases has been omitted, and that is the sarcomata, which should always be thought of in the presence of a tumor when making a diagnosis of tubercular peritonitis.

DR. CAILLÉ.—Tuberculosis and malaria seem to me to be the most important diseases just now, and the nearer we approach the time when tuberculosis will be curable the more I become interested in it. All observations on tuberculosis are worth reporting, and it was with that idea I reported my cases of tubercular peritonitis. I am convinced medicinal treatment in these cases is of no avail; it is not worth while wasting time on guaiacol, creosote and those things. I have tried them carefully and faithfully for a long time on a number of cases, and valuable time is wasted, so that the opening of the abdomen becomes of no importance afterwards.

Now, as regards the diagnosis, I fully agree with Dr. Cotton that we cannot make it, possibly, unless we find the tubercle

bacillus, and that very often is not found. It is claimed that by making a culture from the fluid you will get the tubercle bacillus, if it is there; but at any rate the laboratory men frequently report "no tubercle bacilli found"; and still when we open the abdomen we find tuberculosis. If we suspect the disease and are in doubt, then it is our absolute duty to open the abdomen. You may have a fluid due to chronic malarial infection and an enlarged spleen, or you may have fluid in the abdomen due to cirrhosis of the liver, and other anemic conditions may cause fluid, such as nephritis and valvular heart disease. If you cannot make a diagnosis otherwise, the opening of the abdomen is harmless; it does not put the patient in danger; it is done under an anesthetic, and I think we are derelict in our duty if we do not order it done, for it will absolutely establish the diagnosis. Then, if you are in doubt, the opening of the abdomen is the correct proceeding. As to the value of leucocytosis, I will say leucocytosis may be present in chronic anemia or malaria, and it may aid us in diagnosis, but it is not positive of itself.

PANCREATIC DIGESTION OF CASEIN.

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In the following experiments, which were devised for the purpose of studying certain phases of the pancreatic digestion of casein, I used rabbits' pancreatic juice obtained by the method I have elsewhere described.* Pancreatic juice, thus obtained, was collected in a common receptacle and afterward equally divided between the digestion tubes of an experiment, so that each tube might contain an equal quantity of pancreatic juice of like digestive capacity.

The bile was also obtained from the rabbit and filtered before using. The milk employed was ordinary dairy milk, boiled and neutralized.

Each digestion tube of an experiment contained the same quantity of this milk, diluted either with an equal quantity of water or some other diluent, as detailed in the various experiments.

The digestion tubes were kept in a water bath, at a temperature of 38° C., for five or six hours, and their contents were stirred from time to time with glass rods especially prepared for the purpose. At the close of each experiment the undigested casein in each tube was coagulated by the addition of lactic acid and a saturated solution of ammonium sulphate. By filtration, in a warm chamber, this undigested casein was received on weighed and marked filter paper, which, after being thoroughly washed, was slowly dried and weighed at a temperature of 100° C. The amount of undigested casein in each tube was obtained by subtracting from the gross weight thus obtained the weight of the corresponding filter paper. Tube no. 1 of each experiment contained the same quantity of milk as the other tubes, but did not contain pancreatic juice or other ingredients which might change the casein. At the close of an experiment, therefore, tube no. 1 contained unchanged casein which, when coagulated, was used to determine the amount of casein each tube contained at the beginning of the experiment. The amount of casein which had been converted into peptones in each tube was obtained by subtracting from the amount of

*American Journal of Physiology. Vol. ii., No. 5.

casein in tube no. 1 the amount of undigested casein in each of the subsequent tubes.

It will be noted that 15 cubic centimetres of the different specimens of milk used in the various experiments, did not always contain the same amount of casein, and it is for this reason that the corresponding tubes of different experiments cannot be compared with one another. The comparative accuracy, however, of the deductions drawn from a comparison of the various tubes of an experiment is assured by the fact that the same quantity of the same milk was used in each tube of an experiment.

The maltose solution used in these experiments was prepared by subjecting a mixture of water and one of the Liebig foods to the action of a diastase for one hour. At the end of this time, the diastatic ferment was destroyed by boiling and the maltose solution filtered through ordinary filter paper.

By the above method the following experiments were made:

EXPERIMENT I.—TIME, 6 HOURS.

Contents of Tubes.	Panc. Juice.	Bile.	Undigested Casein.	Digested Casein.	Tube Numbers.
Milk, 15 c c..... Water, 15 c.c.....	o	o	1.174	o	1
Milk, 15 c.c..... Water, 15 c.c.....	m. 6	o	.620	.554	2
Milk, 15 c.c..... Water, 15 c.c.....	m. 6	o	.640	.534	3
Milk, 15 c.c..... Water, 15 c.c.....	m. 6	m. 12	.593	.581	4
Milk, 15 c.c..... Water, 15 c.c.....	m. 6	m. 12	.590	.584	5
Milk, 15 c.c..... Water, 15 c.c..... HCl. Dilute, m. ½.....	m. 6	m. 12	.580	.594	6
Milk, 15 c.c..... Water, 15 c.c..... HCl. Dilute, m. ½.....	m. 6	m. 12	.584	.590	7
Milk, 15 c.c..... Water, 15 c.c..... HCl. Dilute, m. 1.....	m. 6	m. 12	.607	.567	8
Milk, 15 c.c..... .4 per cent. Sol. Sodium Carbonate, 15 c c.....	m. 6	o	.532	.642	9
Milk, 15 c.c.. ..... .8 per cent. Sol. Sodium Carbonate, 15 c.c.....	m 6	o	.521	.653	10
Milk, 15 c.c..... Lime water, 15 c.c.....	m. 6	o	.522	.652	11

EXPERIMENT II.—TIME, 6 HOURS.

Contents of Tubes.	Panc. Juice.	Bile.	Undigested Casein.	Digested Casein.	Tube Numbers.
Milk, 15 c.c. Water, 15 c c.	0	0	1.125	0	1
Milk, 15 c.c. Water, 15 c.c.	m. 5	0	.697	.428	2
Milk, 15 c.c. Lime Water, 15 c.c.	m. 5	0	.640	.485	3
Milk, 15 c.c. Lime Water, 15 c.c.	m. 5	m. 10	.583	.542	4
Milk, 15 c.c. .4 per cent. Sol. Sodium Carbonate, 15 c c.	m. 5	0	.584	.541	5
Milk, 15 c c. .4 per cent. Sol. Sodium Carbonate, 15 c.c.	m. 5	m. 10	.540	.585	6
Milk, 15 c.c. Water, 15 c c. HCl. Dilute, m. ½	m. 5	m. 10	.598	.527	7
Milk, 15 c.c. Water, 15 c.c. HCl. Dilute, m. ½	m. 5	m. 10	.606	.519	8
Milk, 15 c.c. Water, 15 c.c. HCl. Dilute, m. 1	m. 5	m. 10	.660	.465	9
Milk, 15 c.c 2 per cent. Sol. of Milk Sugar, 15 c.c.	m. 5	m. 10	.651	474	10
Milk, 15 c.c. Maltose Solution, 15 c.c.	m. 5	m. 10	.690	435	11
Milk, 15 c.c. Maltose Solution, 15 c.c.	m. 5	m. 10	.639	.486	12

EXPERIMENT III.—TIME, 5 HOURS.

Contents of Tubes.	Panc. Juice.	Bile.	Undigested Casein.	Digested Casein.	Tube Numbers.
Milk, 15 c.c. Water, 15 c.c.	o	o	1.039	o	1
Milk, 15 c.c. Water, 15 c.c.	m. 6	m. 8	.581	.458	2
Milk, 15 c.c. Water, 15 c.c. HCl. Dilute, m. ½	m. 6	m. 8	.531	.508	3
Milk, 15 c.c. Water, 15 c.c. HCl. Dilute, m. 1	m. 6	m. 8	.579	.460	4
Milk, 15 c.c. Water, 15 c.c. HCl. Dilute, m. ½.	m. 6	m. 8	.495	.544	5
Milk, 15 c.c. Water, 15 c.c. HCl. Dilute, m. 1	m. 6	m. 8	.629	.410	6

EXPERIMENT IV.—TIME, 5 HOURS.

Contents of Tubes.	Panc. Juice.	Bile.	Undigested Casein.	Digested Casein.	Tube Numbers.
Milk, 15 c c. Water, 15 c.c.	o	o	1.115	o	1
Milk, 15 c.c. Water, 15 c.c.	m. 10	o	.425	.690	2
Milk, 15 c.c. Water, 15 c.c. HCl. Dilute, m. ½	m. 10	o	.435	.680	3
Milk, 15 c.c. Water, 15 c.c. HCl. Dilute, m. 1	m. 10	o	.588	.527	4
Milk, 15 c.c. Water, 15 c.c. HCl. Dilute, m. ½	m. 10	o	.518	597	5
Milk, 15 c.c. Water, 15 c.c. HCl. Dilute, m. 1	m. 10	o	.596	.519	6
Milk, 15 c.c. 4 per cent. Sol. Sodium Carbonate, 15 c.c.	m. 10	o	.418	.697	7

EXPERIMENT V.—TIME, 5 HOURS.

Contents of Tubes.	Panc. Juice.	Bile.	Undigested Casein.	Digested Casein.	Tube Numbers.
Milk, 15 c.c. Water, 15 c.c.	0	0	1.080	0	1
Milk, 15 c.c. Water, 15 c.c.	m. 8	m. 10	.570	.510	2
Milk, 15 c.c. Water, 15 c.c. HCl. Dilute, m. ½	m. 8	m. 10	.510	.570	3
Milk, 15 c.c. Water, 15 c.c. HCl. Dilute, m. ½	m. 8	m. 10	.482	.598	4
Milk, 15 c.c. Lime Water, 15 c.c.	m. 8	m. 10	.505	.575	5
Milk, 15 c.c. 2 per cent. Sol. of Milk Sugar, 15 c.c.	m. 8	m. 10	.551	.529	6

EXPERIMENT VI.—TIME, 7 HOURS.

Contents of Tubes.	Panc. Juice.	Bile.	Undigested Casein.	Digested Casein.	Tube Numbers.
Milk, 20 c c. Water, 20 c.c.	0	0	1.705	0	1
Milk, 20 c.c. Water, 20 c.c.	m. 8	m. 10	1.035	.670	2
Milk, 20 c.c. Water, 20 c.c. HCl. Dilute, m. ½	m. 8	m. 10	.889	.816	3
Milk, 20 c.c. Water, 20 c.c. HCl. Dilute, m. ½	m. 8	m. 10	1.005	.690	4
Milk, 20 c.c. Maltose Solution, 20 c.c.	m. 8	m. 10	.905	.790	5

EXPERIMENT VII.—TIME, 5 HOURS.

Contents of Tubes.	Panc. Juice.	Bile.	Undigested Casein.	Digested Casein.	Tube Numbers.
Milk, 15 c.c. Water, 15 c.c.	0	0	1.060	0	1
Milk, 15 c.c. Water, 15 c.c.	m. 8	m. 10	.640	.420	2
Milk, 15 c.c. Water, 15 c.c. HCl. Dilute, m. ½	m. 8	m. 10	.579	.481	3
Milk, 15 c.c. Water, 15 c.c. HCl. Dilute, m. ½	m. 8	m. 10	.560	.500	4
Milk, 15 c.c. Lime Water, 15 c.c.	m. 8	m. 10	.609	.451	5
Milk, 15 c.c. Maltose Solution, 15 c.c.	m. 8	m. 10	.639	.421	6

EXPERIMENT VIII.—TIME, 5 HOURS.

Contents of Tubes.	Panc. Juice.	Bile.	Undigested Casein.	Digested Casein.	Tube Numbers.
Milk, 15 c.c. Water, 15 c c.	0	0	1.076	0	1
Milk, 15 c.c. Water, 15 c.c.	m. 8	m. 10	.569	.507	2
Milk, 15 c.c. Water, 15 c c. HCl. Dilute, m. ½	m. 8	m. 10	.536	.540	3
Milk, 15 c.c. Water, 15 c.c. HCl. Dilute, m. ½	m. 8	m. 10	.529	.547	4
Milk, 15 c.c. Lime Water, 15 c.c	m. 8	m. 10	.503	.573	5
Milk, 15 c.c. Maltose Solution, 15 c.c.	m. 8	m. 10	.556	.520	6

In order to facilitate the study of the questions involved in the above experiments, I have, by grouping the tubes bearing upon the same subject, made a number of tables which will now be considered under appropriate headings.

INFLUENCE OF MALTOSE ON THE PANCREATIC DIGESTION OF CASEIN.

TABLE I.

Contents of Tubes.	Panc. Juice.	Bile.	Amount of Casein Digested.
Milk, 15 c.c.+Water, 15 c.c.....	m. 8	m. 10	.420
Milk, 15 c.c.+Maltose Sol., 15 c.c.....	m. 8	m. 10	.421
Milk, 15 c.c.+Water, 15 c.c.....	m. 8	m. 10	.507
Milk, 15 c.c.+Maltose Sol., 15 c.c.....	m. 8	m. 10	.520
Milk, 15 c.c +Water, 15 c.c.....	m. 8	m. 10	.670
Milk, 15 c.c.+Maltose Sol., 15 c.c.....	m. 8	m. 10	.790
Milk, 15 c.c.+Water, 15 c.c.....	m. 5	m. 10	.428
Milk, 15 c.c.+Maltose Sol., 15 c.c.....	m. 5	m. 10	.435
Milk, 15 c.c.+Water, 15 c.c.....	m. 5	m. 10	.428
Milk, 15 c.c.+Maltose Sol., 15 c.c.....	m. 5	m. 10	.486
Milk, 15 c.c.+Water, 15 c.c.....	m. 8	m. 10	.510
Milk, 15 c.c.+Milk Sugar Sol., 15 c.c.....	m. 8	m. 10	.529
Milk, 15 c.c.+Water, 15 c.c.....	m. 5	m. 10	.428
Milk, 15 c.c.+Milk Sugar Sol., 15 c.c.....	m. 5	m. 10	.474

By a study of this table it will be noted that the pancreatic digestion of casein was in every instance slightly facilitated by the presence of a maltose solution, and that in experiments nos. 6 and 7 of this series, a milk sugar solution seemed to exercise the same favorable influence. The inference, therefore, from this table is that rabbits' pancreatic juice in the presence of bile is somewhat assisted in casein proteolysis by the presence of a maltose or a milk sugar solution.

In a previous paper* I demonstrated the physiological fact that acid proteids, undergoing digestion, will slightly increase the diastatic action of rabbits' pancreatic juice. It would seem, therefore, from these observations that the inference may be drawn that both the diastatic and proteolytic action of rabbits' pancreatic juice goes on more rapidly when the juice is acting upon a mixture of starches and albumens, than when the juice is acting separately upon these food stuffs.

* *American Journal of Physiology.* Vol. ii., No. 5.

It must, however, be remembered that there are some difficulties in the way of applying these principles in the solution of the much discussed question of the value of gruels in infant feeding.

Jacobi has long taught that, in healthy children, milk digestion goes on more satisfactorily when it is mixed with a decoction of one of the cereals; and most of the recent writers upon the subject of children's feeding have come to agree with Jacobi, believing, as they do, that under the influence of these decoctions the rennet and hydrochloric acid of the stomach precipitate the casein in more flocculent clots, thus enabling the ferments to come in more intimate contact with the casein to be digested.* Whatever may be the explanation, however, I think we may possibly infer from the above experiments that the favorable influence of these cereal decoctions on casein digestion is continued even after the milk leaves the stomach and comes under the influence of the various digestive enzymes of pancreatic juice in the intestinal canal.

INFLUENCE OF LIME WATER ON THE PANCREATIC DIGESTION OF CASEIN.

TABLE II.

Contents of Tubes.	Panc. Juice.	Bile.	Amount of Casein Digested.
Milk, 15 c.c.+Water, 15 c.c.....	m. 6	o	.554
Milk, 15 c.c.+Lime Water, 15 c.c.....	m. 6	o	.652
Milk, 15 c.c. +Water, 15 c.c.....	m. 5	m. 10	.428
Milk, 15 c.c.+Lime Water, 15 c.c.....	m. 5	o	.405
Milk, 15 c.c.+Water, 15 c.c.....	m. 5	m. 10	.428
Milk, 15 c.c.+Lime Water, 15 c.c.....	m. 5	m. 10	.542
Milk, 15 c.c.+Water, 15 c.c.....	m. 8	m. 10	.510
Milk, 15 c.c.+Lime Water, 15 c.c..	m. 8	m. 10	.575
Milk, 15 c.c.+Water, 15 c.c.....	m. 8	m. 10	.420
Milk, 15 c.c.+Lime Water, 15 c.c.....	m. 8	m. 10	.451
Milk, 15 c.c.+Water, 15 c.c..... ...	m. 8	m. 10	.507
Milk, 15 c.c.+Lime Water, 15 c.c.....	m. 8	m. 10	.573

A study of this table indicates that lime water slightly increases the proteolytic action of rabbits' pancreatic juice on casein. The important role which lime water has long played

* Chapin, ARCHIVES OF PEDIATRICS, December, 1899.

in the milk feeding of infants has given it, in certain conditions, an empirical value which cannot be doubted. It is perhaps true that the beneficial results which are obtained from the use of lime water, in the gastric digestion of milk, are in part due, as Dr. Chapin said in a paper before this Society last year, to the fact that the action of rennet is facilitated by the presence of the salts of lime. It also, however, has some value in neutralizing the acidity which has almost always developed in dairy milk before it has reached the dwelling houses in our large cities. And may it not also be possible that the beneficial influence of lime water on the pancreatic digestion of casein is exerted in somewhat the same way? That is to say, in the milk feeding of infants the lime water, by facilitating the flaky deposit of casein in the stomach, causes the casein to come into the presence of the pancreatic juice in a more suitable form for active proteolysis, and it may even be conceived that the lime salts themselves may reach the intestine, there to stimulate the pancreatic digestion of casein.

INFLUENCE OF SODIUM CARBONATE ON THE PANCREATIC DIGESTION OF CASEIN.

TABLE III.

Contents of Tubes.	Panc. Juice.	Bile.	Amount of Casein Digested.
Milk, 15 c.c.+Water, 15 c.c.....	m. 6	o	.554
Milk, 15 c.c.+.4 per cent. Sod. Carb. Sol., 15 c.c.....	m. 6	o	.642
Milk, 15 c.c.+Water, 15 c.c	m. 5	m. 10	.428
Milk, 15 c.c.+.4 per cent. Sod. Carb. Sol., 15 c.c..... ...	m. 5	o	.541
Milk, 15 c.c.+Water, 15 c.c.....	m. 5	m. 10	.428
Milk, 15 c.c +.4 per cent. Sod. Carb. Sod., 15 c.c.....	m. 5	m. 10	.585
Milk, 15 c.c.+Water, 15 c.c.....	m. 10	o	.690
Milk, 15 c.c.+.4 per cent. Sod. Carb. Sol., 15 c.c	m. 10	o	.697
Milk, 15 c.c.+Water, 15 c.c	m. 6	o	.554
Milk, 15 c.c.+.8 per cent. Sod. Carb. Sol., 15 c.c.....	m. 6	o	.653

The study of this table shows that the presence of sodium carbonate greatly increases the proteolytic action of rabbits' pancreatic juice on casein. The physiological observation is of

importance because of the fact that sodium carbonate is a normal constituent of the succus entericus. One may infer, therefore, that the alkaline intestinal juice will facilitate the action of trypsin on casein. One cannot, however, say, that the value of sodium carbonate in the milk feeding of children depends either wholly or partly upon this physiological fact, since it is quite impossible to see how sodium carbonate could pass through the acid contents of the stomach and reach the intestinal canal in a condition to facilitate the pancreatic digestion of casein. The good that comes from sodium carbonate in infant feeding is probably due to the fact that it neutralizes the fermentation acids which have been formed in the milk.

INFLUENCE OF COMBINED HYDROCHLORIC ACID ON THE PANCREATIC DIGESTION OF CASEIN.

TABLE IV.

Contents of Tubes.	Panc. Juice.	Hydrochl. Acid Dilute.	Amount of Casein Digested.
Milk, 15 c.c.+Water, 15 c c.....	m. 10	0	.690
Milk, 15 c.c.+Water, 15 c.c.....	m. 10	m. ½	.680
Milk, 15 c.c.+Water, 15 c.c.....	m. 10	0	.690
Milk, 15 c.c.+Water, 15 c.c.....	m. 10	m. ½	.597
Milk, 15 c.c.+Water, 15 c c.....	m. 10	0	.690
Milk, 15 c.c.+Water, 15 c.c.....	m. 10	m. 1	.527
Milk, 15 c.c.+Water, 15 c.c.....	m. 10	0	.690
Milk, 15 c.c.+Water, 15 c.c.....	m. 10	m. 1	.519

The few experiments recorded in this table indicate that combined hydrochloric acid slightly retards the proteolytic action of trypsin on casein. The retarding influence, however, is not very great, with the amount of acid here used, considerable proteolysis being accomplished by the pancreatic juice when one minim of dilute hydrochloric acid was added to fifteen cubic centimetres of milk.

INFLUENCE OF BILE AND COMBINED HYDROCHLORIC ACID ON THE PANCREATIC DIGESTION OF CASEIN.

TABLE V.

Contents of Tubes.	Panc. Juice.	Bile.	Hydrochl. Acid Dilute.	Amount of Casein. Digested.
Milk, 15 c.c.+Water, 15 c.c.....	m. 6	m. 12	o	.581
Milk, 15 c.c.+Water, 15 c.c.....	m. 6	m. 12	m. ½	.594
Milk, 15 c.c.+Water, 15 c.c.....	m. 6	m. 12	o	.581
Milk, 15 c.c.+Water, 15 c.c.....	m. 6	m. 12	m. ½	.590
Milk, 15 c.c.+Water, 15 c.c.	m. 5	m. 10	o	.428
Milk, 15 c c.+Water, 15 c.c.....	m. 5	m. 10	m. ½	.527
Milk, 15 c.c.+Water, 15 c.c.....	m. 5	m. 10	o	.428
Milk, 15 c.c.+Water, 15 c.c.....	m. 5	m. 10	m. ½	.519
Milk, 15 c.c.+Water, 15 c.c.....	m. 6	m. 8	o	.458
Milk, 15 c c.+Water, 15 c.c.....	m. 6	m. 8	m. ½	.508
Milk, 15 c.c.+Water, 15 c.c.....	m. 6	m. 8	o	.458
Milk, 15 c.c.+Water, 15 c.c.....	m. 6	m. 8	m. ½	.544
Milk, 15 c.c.+Water, 15 c.c	m. 8	m. 10	o	.510
Milk, 15 c.c.+Water, 15 c.c.....	m. 8	m. 10	m. ½	.570
Milk, 15 c.c.+Water, 15 c.c.....	m. 8	m. 10	o	.510
Milk, 15 c.c.+Water, 15 c.c.....	m. 8	m. 10	m. ½	.598
Milk, 15 c.c.+Water, 15 c.c.....	m. 8	m. 10	o	.670
Milk, 15 c.c +Water, 15 c.c	m. 8	m. 10	m. ½	.690
Milk, 15 c.c.+Water, 15 c.c.....	m. 8	m. 10	o	.670
Milk, 15 c.c.+Water, 15 c.c.....	m. 8	m. 10	m. ½	.816
Milk, 15 c.c.+Water, 15 c.c.....	m. 8	m. 10	o	.420
Milk, 15 c.c.+Water, 15 c.c	m. 8	m. 10	m. ½	.481
Milk, 15 c.c.+Water, 15 c.c.....	m. 8	m. 10	o	.420
Milk, 15 c.c.+Water, 15 c.c.....	m. 8	m. 10	m ½	.500
Milk, 15 c.c.+Water, 15 c.c.....	m. 8	m. 10	o	.507
Milk, 15 c.c.+Water, 15 c.c	m. 8	m. 10	m. ½	.540
Milk, 15 c.c.+Water, 15 c.c.....	m. 8	m. 10	o	.507
Milk, 15 c.c.+Water, 15 c.c.....	m. 8	m. 10	m. ½	.547
Milk, 15 c.c.+Water, 15 c.c.....	m. 6	m. 12	o	.584
Milk, 15 c.c.+Water, 15 c.c	m. 6	m. 12	m 1	.567
Milk, 15 c.c.+Water, 15 c.c.....	m. 5	m. 10	o	.428
Milk, 15 c.c.+Water, 15 c.c.....	m. 5	m. 10	m. 1	.465
Milk, 15 c.c.+Water, 15 c.c.....	m. 6	m. 8	o	.458
Milk, 15 c.c.+Water, 15 c.c.....	m. 6	m. 8	m. 1	.460
Milk, 15 c.c.+Water, 15 c.c.....	m. 6	m. 8	o	.458
Milk, 15 c.c.+Water, 15 c.c.....	m. 6	m. 8	m. 1	.410

If one refers to experiment no. 1 in connection with the study of this table, it is evident that bile not only neutralizes the retarding influence of combined hydrochloric acid on the pan-

creatic digestion of casein, but that by its presence it enables the pancreatic juice to do more work on acid casein than it could do on neutral casein, or on neutral casein mixed with bile. That is to say, bile assists the pancreatic juice in the digestion of casein, but it renders even greater assistance when the casein is partly saturated with hydrochloric acid. When, therefore, rabbits' bile and rabbits' pancreatic juice are brought in contact with acid casein, conditions are provided which favor the proteolytic action of trypsin on casein.

This table shows that the addition of a small percentage of hydrochloric acid almost invariably increases the proteolytic action of pancreatic juice upon casein, when the juice is acting in the presence of bile. And when one remembers that in the carnivora the duodenal contents are always acid, and that even in the herbivora a certain amount of hydrochloric acid is combined with proteids as they are discharged from the stomach into the duodenum, and that the intestinal contents lose their acidity and become alkaline in their reaction, only after they have passed down some distance from the pylorus (in the carnivora a longer distance than in the herbivora), then one can see the force of the above physiological propositions in explaining the digestion of milk in the intestinal canal of all animals, including man. In the infant of the human species, for example, let us suppose that the milk, after being subjected in the stomach to the influence of rennet, hydrochloric acid and pepsin, is discharged, partially digested, through the pylorus, into the duodenum; the casein being either wholly or partly saturated with hydrochloric acid is brought at once under the influence of a mixture of bile and pancreatic juice, and these conditions, as we have demonstrated, being most favorable to the pancreatic digestion of casein, proteolysis will go on rapidly. As the casein passes down the intestinal canal, it presently finds itself in an alkaline medium, the combined hydrochloric acid being wholly neutralized by the sodium carbonate and other alkalies found in the intestinal juices. In this alkaline medium, as we demonstrated in table no. 3, the trypsin still finds itself under conditions most favorable to its action, and proteolysis thus continues under favorable influences throughout the intestinal canal.

That a small amount of combined hydrochloric acid will, in the presence of bile, actually assist the proteolytic action of pancreatic juice on casein, is a physiological fact which has some bearing on the feeding of sick infants.

Jacobi, in speaking of infant feeding, says: "In acute and debilitating diseases which furnish no, or little, hydrochloric acid in the gastric secretion, a small quantity of the latter, well diluted, must be provided for." This is but one of many expressions I find, noting the value of hydrochloric acid in the feeding of sick children. In recent years my own clinical experience has taught me that hydrochloric acid is one of the most valuable agents we have in the treatment of diseases marked by feeble digestion in infants. Hydrochloric acid is, I believe, of special value, as Jacobi says, in those cases where malnutrition is pronounced, and the hydrochloric acid of the gastric juice is for this reason deficient. I have found it of value, however, in almost all cases where there is deficient casein digestion, as manifested by curds in the stools. Casein dilution, as Rotch has so clearly demonstrated, is the rational treatment of this condition. Yet if we are to look to the proper nutrition of the infant, there is a limit to the amount of dilution which may be resorted to. In these cases I have often obtained the greatest benefit from the use of a pepsin hydrochloric acid solution. In my hospital wards I have used this mixture with great satisfaction in infants suffering from casein indigestion, due wholly or partly to a general malnutrition. These cases, as a rule, respond quickly to the acid, the curds diminish or disappear from the stool, and the infant is able to take and digest more milk. I wish, however, especially to note that the good effects of hydrochloric acid are not limited to these cases of malnutrition, but that it is also of real value in almost all cases of casein indigestion, whatever may be the cause, and whether the infant is being fed on breast milk, or some dilution of cow's milk.

In the light of the above experiments we can see that the beneficial action of hydrochloric acid is not confined to the stomach, but as combined hydrochloric acid it is continued in the intestinal canal, where it not only aids the pancreatic digestion of casein but also acts as an intestinal antiseptic. It is my belief that a small portion of hydrochloric acid combined with proteids will, under certain conditions, aid the action of the enzymes of pancreatic juice, while at the same time it exercises a restraining influence on fermentations carried on by organized ferments.

In closing this paper I wish to add a note on certain changes

which take place in the cream of milk when subjected to the combined influence of bile and pancreatic juice.

In the experiments above recorded I noted at the close of certain experiments, that free fat, or butter, was found floating on the surface of all those digestive mixtures, in which the milk had been subjected to the action of both bile and pancreatic juice. In other words, it was noted that the physiological emulsion of fats, as it occurs in milk, was partially destroyed by the combined action of bile and pancreatic juice, but that this emulsion was not destroyed by the action of either one of these agents when acting alone. This observation suggests the possibility that the emulsion of fats in milk is wholly or partially destroyed by the action of bile and pancreatic juice in the intestinal canal prior to their absorption. If it be true that the milk emulsion is destroyed in the intestinal canal, and the fats set free, we can readily understand how in certain diseases of the intestinal canal, which interfere with the absorption of foods, we may have, even in milk-fed infants, greasy or fatty stools.

I wish again in this paper, as I have in previous ones, to acknowledge the skilful assistance of Dr. F. A. Southgate. I am also indebted to Dr. Dudley Webb for valuable assistance.

323 BROADWAY.

DISCUSSION.

DR. ROTCH.—I do not quite understand which Dr. Rachford considered the more valuable, lime water or the cereals, or how they compare.

DR. RACHFORD.—You can see by the table that the cereal has very little influence, but what influence it has is favorable. It has not as much influence as the lime water or as carbonate of soda, but it has no retarding action and in every instance there is a slight increase in proteolytic action.

DR. ROTCH.—I was wondering whether that would not be a reason for using the lime water for the proteolytic action, rather than the cereals, since whenever the cereals are used the amylolytic action has to be brought into play; and whether it is not better to use the lime water than the cereal, as there is so little difference between the two, the cereal giving an extra burden for the digestion of the infant.

DR. RACHFORD.—May I not ask Dr. Rotch whether he has ever seen greasy or fat stools?

DR. ROTCH.—Yes; there is often fat excreted beyond what is needed for nutrition. It seems to depend more upon the extra fat taken in the food and various other reasons of which we do not know. It does not seem to be an abnormal condition.

DR. CAILLÉ.—I was very much pleased to hear Dr. Rachford bring attention to hydrochloric acid in the aid of digestion. Ever since we have had investigation of the stomach contents I have administered hydrochloric acid, and of the few drugs I do give it is one of the few I prescribe daily. I know of no drug that compares with it. From a practical standpoint, therefore, it has been satisfactory to me, and the theoretical reasoning given by Dr. Rachford simply will aid us in understanding why it should be such a satisfactory addition to our pharmacopeia. Now there is one practical point in its administration upon which I would like to lay stress and that is this: In malnutrition or any digestive disturbance whatsoever, the coated tongue is an indication for the administration of hydrochloric acid, and then as soon as the tongue clears up, if any tonic is indicated you may stop the hydrochloric acid and give iron or something else. The coated tongue, therefore, is my indication for hydrochloric acid, and the clearing up of the tongue is my indication for stopping it.

DR. FRUITNIGHT.—I have used hydrochloric acid for a number of years, and where there are lumpy stools I use pepsin and hydrochloric acid. I usually administer them after the meal, sometimes before; they go into the stomach at the same time as the meal. After many years' use of hydrochloric acid I esteem it highly.

DR. BLACKADER.—I would like to ask Dr. Rachford whether fat is usually manifested in the stools in the form of greasy stools. In my own work I usually recognize as fat the light yellow masses resembling casein, which are soluble in ether, or mostly soluble. The fat in the stools giving the stools a greasy appearance I have very seldom seen. The fat, I have been taught, appears in the stools as flocculent masses. Is not it possible to recognize fat only by chemical examination?

DR. HOLT.—I must confess to have seen little benefit from the drugs that have been mentioned. I have come to the conclusion that the treatment is largely dietetic, and by stomach washing we can do more than by all other means combined. The findings in the stomach washing and the fact that the hydrochloric acid is diminished helps very little and is very uncertain. I think its use is purely empirical if we consider our clinical results, and I am inclined to question the specific effect we have given to it. The more I treat these cases the more I am convinced that drugs play a very small part. When we decide with what element of food the trouble is most concerned,

then by remedying that, and giving the children plenty of fresh air and good hygiene, we relieve them. The tendency of the general practitioner to dose these cases with hydrochloric acid and pepsin, and ignore the really important thing, seems to me to not give the best results. As a prominent or general principle of action, I must say I have my doubts about the efficacy of these drugs.

DR. MILLER.—I would just emphasize the remarks of the last speaker. I confess I believe the administration of hydrochloric acid either before or after the taking of food is not according to our studies in physiology. Hydrochloric acid I believe is not secreted just after the introduction of milk into the stomach. It seems to me better to wash out the stomach, or when the washing of the stomach is difficult and not easily resorted to, we may administer an alkali to dissolve away the mucus and allow digestion to go on normally, and that I believe is more in accordance with our ideas of therapeutics. I have tried hydrochloric acid in infants and I have, except in a few cases, been disappointed. Still I think we as a Society should not emphasize the use of drugs in a special way, but rather the broad principles, as just laid down by Dr. Holt.

DR. CAILLÉ.—Perhaps the difference in opinion in regard to the value of hydrochloric acid can be explained in this way: sometimes it does not give brilliant results in infants but it does give good results in children above the age of two. The small value ascribed to hydrochloric acid is probably because some have infants under two years in mind.

DR. ROTCH.—My experience has been, in the use of drugs and diet, that the diet is the prime factor in the problem, and the reducing of the proteids, to which Dr. Rachford has referred, is also valuable. When we are using the proteids of cow's milk it seems to me they are somewhat more nourishing than those of human milk and we can get along with a smaller quantity of cow's proteids than of human proteids. It is very seldom, in my judgment, that we will have to use any drug whatsoever.

THE PRESIDENT.—In closing the discussion I wish Dr. Rachford would give us in a few words the reason he did not use potassium carbonate, as has been suggested recently.

DR. RACHFORD.—This whole subject is a very difficult one, and it is especially difficult for a physician engaged in hard work with a busy practice to do, and so the investigation must be quite limited. It is a very tedious process, as it requires a good portion of a week with good assistants to make one of these experiments. The number of subjects experimented upon, therefore, must be limited. I chose sodium carbonate because I wanted to study the normal digestion in the infant if possible, that is, to study the influence the sodium carbonate in the

succus entericus may have on the infant digestion. The idea was to study the influence of rabbit pancreatic juice, and if possible to throw some light on the pancreatic digestion of casein. Whether this will help in children's feeding is another thing. I did not make the research to explain by any means all the things in connection with infant feeding, but I simply desired to shed some light upon the pancreatic digestion of casein.

The observation which I made in the last paragraph of the paper as to the influence which pancreatic juice and bile have on the emulsion of fats interested me, as I said, because it suggested that the emulsion was destroyed and the fats were absorbed in this form as fatty acids coming in contact with alkalies, just as other fats are absorbed, and that the fat probably is not absorbed in the form of an emulsion. I suggested that this might explain the presence of fat in the stools. Occasionally I have seen stools that to the naked eye did not appear as if they contained free fat, but they presented a greasy appearance. In the great mass of cases we may judge from the little yellow, creamy or butter-like masses that the stools are fatty, and then by dissolving out the fat with ether we may determine both the presence and the amount of fat in the stools. And so we often have fat in the stools in the form of butter-fat rather than of cream. The globules are not surrounded by the albuminous envelopes.

Dr. Holt observed that when hydrochloric acid was used it was entirely empirical, and the explanation of its value must be simply an empirical explanation. After making my experiments on the influence of bile and hydrochloric acid on casein, which experiments were devised for the purpose of studying the digestion of casein in the small intestine, I came to the conclusion that combined hydrochloric acid in the presence of bile actually increases the proteolytic action of pancreatic juice on casein. If that is true, and if these experiments are confirmed, then the use of hydrochloric acid in certain conditions will not be empirical, and we will have one reason for the use of hydrochloric acid which we have not had before. However, up to the present time the use of hydrochloric acid has been empirical. In certain conditions there is an absence of hydrochloric acid in the stomach—in cases of malnutrition. We know hydrochloric acid is present in the stomach for a purpose, and the clinical experience of men, who have had really very great clinical opportunities, leads them to believe that hydrochloric acid is of real value in the treatment of this class of cases. And so we may draw conclusions from this that would not be altogether empirical. The absence of hydrochloric acid from the stomach would call for the use of hydrochloric acid, and not entirely in an empirical way. We probably all agree with Dr. Holt that hydrochloric acid is not the most important thing in the treat-

ment of these cases; in fact, it is rather an unimportant thing. The washing out of the stomach and the dietetic treatment of the cases are more important, but here is an additional item in treatment. The use of hydrochloric acid is not to take the place of the other measures, which are really the ones most relied upon, such as cleanliness and proper food and those things referred to in our President's address. Dr. Blackader stated that hydrochloric acid is not indicated, given either before or after meals, because hydrochloric acid is not secreted immediately after the fluid is taken into the stomach. I do not see the force of that argument. Hydrochloric acid is found in the stomach half an hour after the introduction of food, and it is there for a purpose. When hydrochloric acid is absent from the stomach, then we have a reason for giving hydrochloric acid, whether before or during or after the meal. Hydrochloric acid deters the lactic and other fermentations, and there seems to be no reason for waiting half an hour or so to give hydrochloric acid just because it is not normally found until half an hour or so after the ingestion of food. The paper was not prepared for the purpose of extolling the virtue of hydrochloric acid, but to detail certain experiments which I hope will be of value in the treatment of sick children.

A CASE OF RHACHISCHISIS.

BY T. M. ROTCH, M.D.,

Boston, Mass.

Rhachischisis is one of the principal forms of congenital defects of the spine. It is characterized by a deficiency of the vertebral arches either complete or partial. The cord is rudimentary and is split open so that the endothelial lining of the central canal is exposed. This may occur in the whole of the cord or in part of it. The condition is of interest pathologically rather than clinically. The following cases of rhachischisis came under my notice at the Infants' Hospital.

A girl three days old was admitted to the hospital on February 14, 1900, in the service of Dr. John Dane. The history of the case was that the delivery had been with forceps, that the infant was viable, and that it had taken no food since birth. A physical examination showed the head to be of normal size. The anterior fontanelle was widely open. There was a caput succedaneum on both sides. The face was flattened, the chin was retracted and was held in forced position with the occiput resting on the upper dorsal spine as represented in Fig. I.

The front of the neck was bulging. The chest and abdomen were apparently normal. On examining the posterior surface of the infant a cleft was found in the posterior vertebral arches extending from the first dorsal to the third lumbar vertebra, measuring one and a half inches in the middle and narrowing evenly on both sides. The intervening space in its lower part was covered with good skin. Above this in place of the skin there was a parchment like membrane, and an area in the upper part of the cleft one and a half inches long and covered with granulations. No bulging or sense of fluctuation was detected in this cleft. Fig. II. shows this posterior aspect of the infant just described.

The extremities were normal, but were held rigidly with a spasm of all the muscles which, although it could be overcome by slight force, quickly returned. The patellar reflexes were absent. The eyes were open nearly all the time. At long intervals the infant gave a slight cry. It was unable to swallow. The temperature was subnormal. An ophthalmoscopic examination showed both eyes to be normal. On February 18th, four days after entrance, and seven days from the time of birth, the infant died without any especial symptoms.

Before giving a description of a vertical section of the head and thorax made in this case, a few words regarding rhachischisis in general may be of interest. Dr. Augustus Thorndike has written and published such an excellent article* on rhachis-

Fig. I.—FLATTENED FACE AND RETRACTED CHIN IN A CASE OF RHACHISCHISIS

chisis that little is to be said of the condition outside of what he has described, and I shall therefore freely quote from his description. I am also indebted to him for the solar prints of a number of cases of rhachischisis selected from twenty skeletons of this condition which are in the Warren Medical Museum connected with the Harvard Medical School in Boston.

This defect of the central nervous system is due to an arrest of development, or a persistence of early embryonic conditions, and to certain later processes, such as the distension of cavities by transuded fluid (Thorndike). It is often associated with spina bifida, which is distinguished from rhachischisis anatomically by the fact that in spina bifida the spinal

* Transactions American Orthopedic Association, 1899.

cord has been properly formed. Rhachischisis has its counterpart in the skull and brain which are represented by anencephalus (complete absence of the brain), or derencephalus, which corresponds to partial rhachischisis. The condition of total rhachischisis occurs much more frequently with anencephalus than without, and especially occurs in the still-born, or those that live a very short period. In many cases the infant is premature.

TOTAL RHACHISCHISIS.—Fig. III. represents anencephalus with total rhachischisis and shows the short neck, upturned face, and

Fig. II.—POSTERIOR VIEW SHOWING SURFACE COVERED WITH GRANULATIONS.

mane-like growth of hair on the skin of the nape and upper back.

Here the cranial vault is absent, and the base of the skull and the centre of the back is covered by an irregular band of dark red tissue looking like mucous membrane. This is what is called the area medullo-vasculosa. Outside of this is the area epitheliodea shading off into the true skin. Outside of this area epitheliodea can be seen a series of small elevations caused by the ends of the cleft arches under the skin. There is also a narrow line of long fine hair extending about half way down the back and looking like a divided mane.

A longitudinal section made from an eight months' fetus

with total rhachischisis and anencephalus with a posterior encephalocele is represented in Fig. IV. and shows the abnormal curves of the spine which occur in these cases.

These abnormal curvatures, both antero-posterior and lateral, are very common. In this case a microscopic examination made by Dr. Thorndike is described by him as follows:

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Fig. III.—ANENCEPHALUS WITH TOTAL RHACHISCHISIS, SHOWING THE SHORT NECK, UPTURNED FACE, AND MANE-LIKE GROWTH OF HAIR ON THE SKIN OF NAPE AND UPPER NECK.
(*A. Thorndike.*)

It showed the area medullo-vasculosa to be made up of large sized blood-vessels, nerve fibres, neuroglia tissue, and a few ganglion cells. The superficial covering is an epithelial layer of cells, the endothelium of the central canal; from the ventral side the spinal roots pass to the dura and the foramina,

and there was a covering of vascular pia mater to the nerve tissue on its ventral side; there was also a glistening flat membrane on which it lay, the dura. According to Thorndike, the vertebral column frequently presents other deformities besides absence of laminæ and spines.

Fig. IV.—LONGITUDINAL SECTION THROUGH EIGHT-MONTHS' FETUS WITH TOTAL RHACHISCHISIS, SHOWING ABNORMAL CURVES OF SPINE.
(*A. Thorndike.*)

In referring again to Fig. I it will be noticed how in these cases we find a very short back and almost no neck, the ears frequently touching the shoulders, and the face looking upward, instead of forward, the frog-like appearance described by various observers. This retracted position of the head is caused

by a sudden sharp antero-convex curve taken by the upper dorsal and cervical spine. The condition of anencephalus alone is not sufficient to produce this curve.

Fig. V. shows the great deformity of the thorax from an antero-posterior curvature of the dorsal and cervical spine.

Fig. VI. shows a lateral curve and an abnormal anterior cervical curve in a case of anencephalus and total rhachischisis.

Quoting Thorndike's description again, the spinal cleft is usually symmetrical and is due to absence of the spinal processes and of both laminæ, and sometimes the transverse processes are not formed. Occasionally the bodies are cleft, as well as the arches so that there is an anterior and posterior split in the spine; the two halves of the spine thus divided separate from each other and unite below the cleft.

Fig. V.—ABNORMAL FORWARD CONVEX CURVE OF DORSAL AND CERVICAL SPINE IN THE SKELETON OF A CASE OF TOTAL CRANIO-RHACHISCHISIS. (WARREN MUSEUM.)

(*A. Thorndike.*)

PARTIAL RHACHISCHISIS.—Partial rhachischisis means that the formation of the cord is incomplete. The condition is marked by the interposition of an area medullo-vasculosa where the cord fails to form. It is often associated with other deformities and may occur in the premature and in cases that die soon after birth.

According to Thorndike, the abnormal curves of the vertebral column are present in the specimens of partial rhachischisis, but he has not noted such extreme deformity as in the cases of total cleft preserved in the Warren Museum. Any region of the spine may be affected, and sometimes there may be clefts in two different parts of the spine. The cleft, however, is always said to involve four or five vertebræ, sometimes

more. Thorndike states that not infrequently in rhachischisis the area medullo-vasculosa is divided into two symmetrical halves, and is probably due to a failure of the two lateral halves of the undeveloped cord to unite. This condition is called diastematomyelia. According to Recklinhausen's description of a case of partial rhachischisis (*Virchow's Archiv*, Vol. cv.) the skin is lacking over a circular area in the centre of the back, which is a typical area medullo-vasculosa, lying as a broad, dark-red circle surrounded by a smooth, glistening membranous ring, which in turn merges into the true skin of the back. The same structures are present, as in complete rhachischisis, but at the upper and lower end of the area are slight depressions, the cephalic and caudal poles marking the points where the central canal of the cord begins to be a closed tube above and below. Recklinhausen also describes how often an accumulation of fluid in the meshes of the pia beneath raises this area medullo-vasculosa upon the top of a quasi cyst, where it lies spread out and exposed to the air.

Fig. VI.—ANENCEPHALUS AND TOTAL RHACHISCHISIS. SLIGHT LATERAL CURVATURE AND ABNORMAL ANTERIOR CURVE OF NECK. (WARREN MUSEUM.) (*A. Thorndike.*)

Having considered these forms of rhachischisis in general, I will now continue the description of the case of partial rhachischisis without anencephalus of which I have already given the clinical history.

After death the body was placed in a 10 per cent. solution of formalin and was hardened for three weeks. A median longitudinal section was then made by Dr. Thorndike through the entire body, and the right side of this section is represented in Fig. VII.

The viscera, apart from the nervous system, showed no abnormality beyond distension due to the deformity of the bones. The esophagus was forced to the left, and restricted from pressure of the bodies of the upper dorsal vertebræ. The

Fig. VII.—LONGITUDINAL SECTION OF A CASE OF RHACHISCHISIS.

bladder was widely distended. The cartilaginous body of the atlas was found articulating with the odontoid process, which together with the body was ossified and well developed. The bodies of the remaining cervical vertebræ were found to be fused with some evidence of cartilaginous septa. The axis of

the cervical spine, as shown in the figure, is directed backward and upward at right angles to the direction of the upper dorsal spine. There were twelve dorsal, five lumbar, and six sacral bodies (vertebræ) and a coccyx. The arches were wanting below the eighth dorsal vertebra. The skull was apparently well formed. The foramen magnum was as large as a silver dollar. The section of the brain made by Dr. E. W. Taylor passes nearly through the median portion of the brain which was very poorly hardened. The convolutions were present, and the cerebrum was approximately of normal size. Neither the cerebellum nor pons are to be found in the cranial cavity. The pons was found lying in the widened cervical canal below the foramen magnum, and was connected with the cerebrum by brain substance. Considerably below this point, and lying in front of the area medullo-vasculosa, the cerebellum was found and was connected with the brain by an attenuated peduncle. Beneath the cerebellum and on one side of it lay the cord which lost its identity in the area medullo-vasculosa. Springing from the lower part of this area is the lumbar enlargement of the cord and the nerve roots arising from it.

197 COMMONWEALTH AVE.

A FATAL POST-OTITIC CEREBRAL ABSCESS WITH AMNESIC APHASIA.

BY J. HENRY FRUITNIGHT, M.D.,

New York.

The case to be narrated well illustrates how sometimes the extent and gravity of a pathological lesion is not reflected in the clinical history of the case. It again teaches the lesson how an otitis or an otorrhea neglected or intermittently treated at the outset, does sometimes terminate fatally. It also emphasizes the fact that a portion of the laity do not yet realize the gravity of this affection, and it behooves us to continue our efforts in educating them in its possible dangerous complications. The cases which have turned out as failures, which might have become successes if the conditions present had been better understood, or if interference had been undertaken earlier, are just as instructive, and possibly more so, than cases which have a favorable termination. This is the reason why it is deemed worthy that this case should be put on record.

Fannie W., twelve years old, native of New York City, came under my care, December 10, 1899. In June, 1898, while in the country, she suffered severely from an acute otalgia of the left ear. She consulted a well-known physician of the place, who diagnosticated otitis media with an abscess. He made a paracentesis of the drum of the left ear, letting out quite a large amount of pus. The doctor gave the mother instructions how to proceed in the treatment of the resulting otorrhea. This treatment the mother failed or neglected to continue, though warned by the physician that she must keep it up until the discharge should cease. When I asked her why she had not done so she replied: "She had done it for several weeks and as the discharge did not seem to stop, she had become tired and the child had resisted; and besides it was only a running ear, and a friend of hers had had such a 'running ear' for the past seventeen years, and no harm had come in consequence."

Since then the child had had more or less discharge from the ear, being a period of about eighteen months. Several weeks before I was called, she suffered from attacks of severe frontal headache with nausea and vomiting, for which she was treated by a neighboring physician. During these four weeks she was more or less indisposed, but able to be up and about.

When I first saw the patient her temperature was about 100° F.; pulse, 98; and she complained of frontal headache. There was some prostration; she had occasional chilly sensations and nausea; there was a scanty discharge from the left ear.

The clinical diagnosis was, deep mastoid caries, possibly cerebral abscess and beginning meningitis. I at once suggested a consultation, telling her friends that an operation was necessary, but this was refused at the time because the mother considered the affection a trifling one.

On the evening of December 17th, she was seized with violent convulsions, which continued for six hours. They were finally controlled by the use of rectal injections of chloral hydrate, inhalations of chloroform and hypodermic injections of morphin. The prognosis was most unfavorable, but to my surprise the patient still lived on the following morning. I then insisted upon a consultation, and in the afternoon of that day, Dr. Herman Knapp, of New York, saw the patient with me. At the time of consultation the patient was found to be excited and very much frightened, but conscious and rational. Her temperature was 101° F. and her pulse 120. The movements and sensibilities were normal.

Dr. Knapp made an ophthalmoscopic examination of the eyes which revealed that the pupils, the backgrounds of the eyes, the sight and the *field of vision* were normal. There was very little secretion in the left ear. Dr. Knapp also made an otoscopic examination of the auditory canal, but its fundus could not be clearly seen. There were no granulations present, nor was there any sagging of the posterior part of the wall. The mastoid was but little swollen and not particularly tender. The most interesting feature present was an optical amnesic aphasia or word-blindness. If an object were placed before her, and she were asked its name, she would become annoyed and say: "I know what it is, but cannot call it by name." When the name of the object was mentioned, she would repeat it at once and correctly. Thus, if a key were held before her, she seemed

perplexed and angered because she could not name it. When told its name, she would at once say: "Oh, yes, a key."

Dr. Knapp made a clinical diagnosis of "deep mastoid and epitympanic caries, epidural and cerebral abscess with beginning meningitis." The relatives were told that surgery alone could be of avail to save the child's life, and an operation should be performed without delay, as he considered the severe convulsions as a last warning. The consent of the relatives was then obtained. She was taken to the New York Ophthalmic and Aural Institute, and was operated upon by Dr. Knapp, at six o'clock in the evening, in the presence of Drs. Jordan, Nolte, myself and several others. Dr. Knapp very kindly furnished me the following description of the operation and also the record of the subsequent history and of the autopsy.

"*Operation:*—After the usual preparation an incision was made down to the bone, from the tip of the mastoid along the insertion of the auricle, as far as the zygomatic ridge. The bone surface, freed from the periosteum with a raspatory, was vascular, more in the lower than in the upper part. The skin lining the posterior and upper meatal walls was dissected from the bone and drawn out and forward with a strip of aseptic gauze passed along the bared bone, into and out of the ear canal. The mastoid, when opened, was found diploic, vascular, and very brittle. The posterior and upper walls of the bony ear-canal were chiselled away and the attic was laid bare. The latter was packed with cholesteatoma masses, which were cleanly removed.

Then the posterior cranial fossa was exposed by chiselling and curetting away all the carious bone that separated it from the body of the mastoid. The dura and the sigmoid sinus, open to view, showed no abnormity. There was neither epidural abscess nor external pachymeningitis.

After this the upper wall of the attic, which was carious, was removed and the dura of the middle cranial fossa exposed in an area 2.5 cm. by 2 mm. The dura was congested and also slightly uneven and dull. Near the posterior-medial corner of the exposed area I noticed in the dura a blackish, round spot of about 3 to 4 cm. in diameter, with a central depression through which I could introduce a probe 4-5 cm. into the brain, without meeting with any resistance or eliciting blood or pus on withdrawal. The latter condition and the late hour of the day determined me to interrupt the operation. The radical tympano-mastoid operation and the opening of both the middle and posterior fossæ having removed the source of the whole disease and relieved the brain from pressure, could be supposed to place the patient beyond immediate danger and in more favora-

ble circulatory and mechanical conditions for amelioration of the symptoms, and might produce a clearer indication of the location of the abscess, the presence of which in the temporo-sphenoidal lobe, to judge from the history, the word-blindness, and the characteristic black, perforated spot of the dura, could be assumed with a probability that was almost a certainty. The wound, therefore, was cleansed with aseptic gauze, the meatal skin-flap split horizontally, and the outer edge of the latter extended by two vertical incisions. The flap was pressed against the wound of the mastoid with sterilized iodoform gauze, the ear bandaged, and the patient put to bed.

December 19th.—Night quiet. Feels better; is rational. Names most objects at sight. Temperature, 101° to 102° F. Pulse, 110 to 120.

December 20th.—Still better. No word-blindness. Is cheerful. Temperature, 99.3° to 101° F. Pulse, 80 to 100.

December 21st.—Fails to name some objects she sees. Is quite rational. Appetite good. Temperature varying from 99.2° F., pulse, 80; to temperature 100.3° F., pulse 118. Dressing of wound changed; smells strongly.

December 22d.—Quite rational. Appetite poor. Nausea and severe headache. Dressing changed. Temperature, 99.3° F., pulse, 100; to temperature 101.3° F., pulse, 120. Morphia.

December 23d.—Sleep interrupted. Nausea. Severe headache. Anorexia. Temperature, 99° F., pulse, 96; to temperature, 100.8° F., pulse, 108. Quite rational. Wound clean.

December 24th.—In the morning, some nausea and vomiting, slight secretion. Severe headache. No appetite. Two A.M., temperature 97.4° F. At 5.30 A.M., temperature, 98.2° F., pulse, 80; 11 A.M., temperature, 98.3° F., pulse, 70; 9 P.M., temperature, 100.2° F., pulse, 60. In the morning, complained greatly of headache. Morphia. In the afternoon, slept soundly and felt pretty well; in the evening, headache and drowsiness.

This being the first day that the elevated temperature, the slow pulse, and the other symptoms had the character of a brain abscess, an operation was decided upon for the next day.

At 9.45 P.M. she suddenly gave a shriek, jumped out of bed, her face grew purple. At 9.50 she stopped breathing; face white; tongue protruded, and foam came from the mouth. Death.

Autopsy (partial only allowed).—Wound clean; skull thin. The dura shows dark venous congestion. Very few adhesions of the dura to the anterior surface of the petrous bone, some also to the occipital lobe. The dura, as far as exposed by the operation, was thickened by granulations. The blackish, centrally perforated patch still well recognizable. After incising the dura, the blackish patch was found agglutinated loosely to the pia. There was no subdural exudation, and the soft membranes showed no conspicuous abnormity. The veins in the

sylvian fissures were much congested, the gyri and sulci darkened, the latter effaced, *i.e.*, only indicated by lines. The first temporal convolution looked tolerably healthy, the second slightly, and the third very much discolored. In the middle part of the temporal convolutions the brain substance was softened to the extent of 8.5 cm. in length and 5 cm. in height. The softening was immediately above the black patch, where the dura was perforated.

The right hemisphere showed no abnormality.

After the brain had been removed in toto, it was divided in the median line. At once a large quantity of thin puriform offensive liquid, with many small particles, flowed out from the third and left lateral ventricles. The walls of the ventricles were finely roughened. Temporally from the lateral ventricle a large abscess cavity was situated, surrounded by a dense white capsule, which was ruptured in front and toward the lateral sinus behind. It contained the same material as the ventricles. The capsule of the abscess was surrounded by a zone of softened brain substance varying from 5 to 15 mm. in breadth. The track of the probe which had been introduced into the brain could not be discovered.

After hardening in ormol the following conditions were ascertained:

Antero-posterior diameter, 185 cm. (seven inches). An abscess cavity occupied the middle of the temporosphenoidal lobe, situated a little more in the anterior part than in the posterior. It was surrounded by a dense, uniform, white capsule, the thickness of which varied from 0.5 mm. to 5 mm. It was perforated in two places: In front, the contents were mixed with the broken-down surrounding tissue; in the posterior medial wall, into the posterior outer cornu, the contents filling the lateral and third ventricles and mixing with the softened cortex of the adjacent posterior part of the temporosphenoidal lobe.

The inner dimensions of the abscess cavity were: Sagittal, 45 cm. (one and three-fourth inches); frontal, 26 mm. (one inch); lateral, 20 mm.

The inner surface of the abscess cavity was smooth, with some depressions here and there; in the neighborhood of the perforations of the capsule it was uneven and softened."

The symptoms certainly seemed to indicate a meningitis rather than an abscess, and the diagnosis offered by me was that of probable meningitis. Even after the operation the clinical history was not characteristic of cerebral abscess, because the usual combination of elevation of temperature and slow pulse usual in such cases was absent, excepting during the last twenty-four hours. It was, therefore, surprising that at the

autopsy neither a meningitis nor epidural abscess, but only a cerebral abscess, was encountered. Very probably the thick capsule explains this. This thick capsule also proves that the abscess might have existed for quite some time, and it also accounts for its long quiescence.

Though this case proves the statement that the optical memory centre is in the temporosphenoidal lobe, it does not show its exact location, because the softened area was too extensive. Dr. Knapp, however, says, after hardening the brain, "That the cortex of the superior temporal convolution was not softened anywhere, and that of the middle convolution softened only at its lower border and in a small patch in its middle part, whereas the third convolution, both on its lower and lateral surfaces was most extensively softened."

Though optical aphasia and hemianopsia are frequently combined, it will be noted this was not so in this case.

Reasoning from the symptoms and course of the disease in the last month of the patient's life, we are justified in saying that the exacerbations of the trouble leading to the death of the patient was the result of the extension of the abscess beyond the boundaries of its capsule. Increasing inflammation produced the symptoms present in meningitis and in cerebral abscess, including the severe convulsions. At this time also, the perforation in the anterior part of the capsule took place, permitting part of the abscess contents to flow into the surrounding brain tissue, and later, the secondary perforation into the ventricles, caused the sudden death.

DISCUSSION.

DR. KNAPP.—This case is remarkable and very instructive. It is rare enough that we can make a diagnosis of brain abscess with certainty, and still rarer that we can know before the operation the location of the abscess. The symptoms in Dr. Fruitnight's case pointed much more to meningitis than to abscess. But the long duration of the disease made it probable that an abscess could have excited meningitis, and an operation might save the child's life. The operation of opening the mastoid extensively showed evidence of the presence of an abscess: namely in the roof of the tympanum. The dura mater was not sufficiently diseased to have caused the trouble; but it

had a small spot with a central perforation, through which I introduced a probe almost two inches into the brain. The probe met with no resistance at all. It went through the tissue, which was soft, having not even the consistence of brain substance, and nothing came forth. This deceptive condition determined me not to go on with the operation. Had I tried to puncture the brain either with an aspirator or a knife, I might have struck the abscess, but it is quite likely that either instrument would have gone the same way as the probe did, and would have stopped or deflected on its way by the dense abscess membrane.

Brain abscess is not frequent. Meningitis is frequent enough, and thrombosis is still more so. Repeatedly have I gone into the brain when the symptoms of abscess prevailed, and the neurologist advised operation, but no abscess was found even at the autopsy. They were cases of meningitis, some even on the convexity of the frontal lobes.

The case reported by Dr. Fruitnight was evidently an old one, and there was considerable softening, but the capsule, which was very dense towards the ear, was broken anteriorly and posteriorly. The perforation in the anterior part evidently was the cause of the convulsions which the child had and from which it recovered rapidly when the operation had relieved the tension of the brain. The day after the operation the child was rational, talked, and recognized objects. On the fourth day she was not so well. On the fifth her condition was changeable, At 5 P. M. I saw her, she slept soundly. We determined to operate again the next day, but at 9 P. M. she suddenly expired from a perforation of the abscess into the lateral ventricle, as was demonstrated by the autopsy which I made the next day.

DR. CHAPIN.—I would like to call the attention of the Society to one of the phases of mastoid trouble in children in which the brain is not affected, but the pus surrounds the tip of the mastoid, burrows anteriorly and is seen in front of the ear. These are most puzzling cases. I saw recently a little boy five years of age. There was tenderness back of both ears. After the incision of the drums the temperature dropped from 103° F. to about 101.5° F., and stayed there a day or so. There was still tenderness, but the temperature in a few days dropped to normal. Later a swelling appeared, but the temperature was still normal. Then I called in an aurist and we decided it was best to operate. We found some little broken down tissue and with an aspirator we found an abscess in the front of the ear. I think it is well to remember these cases and look also in front of the ear in cases in which we have mastoid disease.

DR. FRUITNIGHT.—I would again specially call attention to the absence of low pulse and high temperature, usually encountered in cases of cerebral abscess.

THE TREATMENT OF HYDROCEPHALUS BY CRANIECTOMY.

BY EDWARD P. DAVIS, A.M., M.D.,

Professor of Obstetrics in the Jefferson Medical College; Professor of Obstetrics and Diseases of Infancy in the Philadelphia Polyclinic; Visiting Obstetrician to the Jefferson, Philadelphia and Polyclinic Hospitals, etc.

The patient, whose history is here reported, was an infant aged six months when seen for the first time. The parents had two other children who did poorly during the first six months of life. Before the birth of this child, the mother was in excellent health, and the birth was spontaneous. She nursed the child for three weeks, when she had what was described as "a bilious attack," and the secretion of milk failed. The child was then fed upon starchy foods. It weighed at birth five and a half pounds, and at six months, nine pounds. It was brought for treatment because it was restless, had a poor appetite with dyspepsia and did not seem to be gaining.

On examination, the child's length was 54 cm. The circumference of its chest was 34 cm., and the circumference of the cranium 41 cm. The contour of the cranium was characteristic of intrauterine rachitis, the child's flesh was flabby, and it was restless, fretful and weak. There were no signs of teeth, and the child had a double inguinal hernia. Three months after it was first seen, its length was 55 cm., its chest, 35 cm., and the circumference of the cranium, 43 cm. The child had gained somewhat in weight during the summer at the seashore. Five and a half weeks after this, the circumference of the child's cranium was 47 cm. It was decidedly stupid and evidently suffering from intracranial pressure, although its digestion was much better. Its stupor had alarmed the parents who had also observed the continuous enlargement of the cranium. When the question of treatment was considered, the parents were informed that medicinal treatment offered nothing, but operative treatment gave a possibility of temporary improvement. They accepted the latter, with a full understanding of its risks.

The child was transferred to the Jefferson Hospital, and was seen by Dr. W. W. Keen in consultation. Dr. Keen was willing to drain the right lateral ventricle, although he expressed a guarded opinion in giving a prognosis. The effort was made in this case to insure continuous drainage and not simply to withdraw fluid from the ventricles. Accordingly, the child was anesthetized and under antiseptic precautions, the cranium was trephined at one side of the sagittal suture and in the parietal bone of the right side. This was accomplished without difficulty, although the cranial bone seemed thinned by the pressure within. A sterile silver cannula containing a stylet was then passed through a slit in the membranes downward until the ventricle was reached, when the stylet was withdrawn. A stream of serous fluid immediately followed. Two strands of sterile silk worm gut were then introduced through the cannula and the cannula withdrawn. The tissues were closed as rapidly as possible by suture, leaving the ends of the silk worm gut emerging through the flaps. Pressure was made over the site of drainage by antiseptic dressings. The child collapsed and did not recover, although it lived for some hours after the operation.

The unfavorable feature about the operation was the fact that considerable fluid escaped. There was no hemorrhage nor apparent shock.

The bad result in this case was by no means unexpected, although the circumstances seemed to warrant an attempt to relieve the patient. The literature of the subject does not give reports which would lead one to hope for permanent recovery after the operation. In the face, however, of the anxiety of parents to do all possible for such children, the attempt is justifiable in the absence of any other known method of treatment. The case is reported with the hope of eliciting an expression of the experience and opinion of the members of this Society.

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DISCUSSION.

DR. ROTCH.—I have been very much interested in this class of cases and have had quite an experience with them. In the Infants' Hospital at Boston we have had them for some years, and have had them operated upon. No bad results came from the operation as such. There were a number of cases, I cannot state exactly how many; they were favorable, if any could be favorable, for operation. They were treated with tapping and I had some cases which we kept for weeks with permanent drainage, by tubes introduced into the ventricles, the fluid being withdrawn every day in small amounts so as to make the child comfortable. The results were invariably bad, so that our opinion now in Boston, from what we have observed, is that the operation in no way is curative. It may be, possibly, palliative, making the child more comfortable for the time being, and it is a perfectly proper operation to do if the parents wish to have something done. So far as being curative it is not a success for hydrocephalus, although we do not know of anything better.

DR. DORNING.—I wish to refer to a case of chronic hydrocephalus, in which I withdrew about two ounces of fluid by lumbar puncture. The child improved very markedly. Prior to the puncture the child had frequent convulsions and was very ill. After the puncture the convulsions ceased and the child seemed to improve for about three weeks. The symptoms reappeared after that and, unfortunately, I lost track of the child.

THE PRESIDENT.—I have had records of three cases of hydrocephalus, two of which I had given over to the surgeons to treat. In these two cases an aspirating needle was passed into the anterior fontanelle and the ventricle aspirated under antiseptic precautions. One patient was two years old and the other a little younger. Both cases did well for a while, and then died with the symptoms of stupor and convulsions. Lately I have had one very interesting case treated in my ward by lumbar puncture, a baby seven months old, with marked signs of rickets and with a marked hydrocephalus. The baby developed symptoms resembling those of tubercular meningitis. The child would cry out at night, would be stupid during the day time, and developed convulsions similar to Dr. Dorning's case. We tapped the child repeatedly, each time withdrawing ten to twenty cubic centimetres of fluid. The baby seemed to improve after each tapping. The convulsions ceased, the baby took its food and noticed us as we made the rounds. The cry at night ceased for a time. We tapped the child six or seven times at intervals of a week with very good results, intending by the tapping to gradually reduce the amount of fluid in the ventricle. A few days after the last tapping the child developed a slight rash

and a temperature of 103°. The temperature ran up to 108° F. and the child died with Cheyne-Stokes respiration and convulsions, although the greatest care was taken with this tapping and the same quantity withdrawn as on the previous occasion, which had been at an interval of about a week. The question arises whether the diminution of pressure had not precipitated the fatal result. An autopsy was made. I suspected there had been a tubercular element, as has been described, and hydrocephalus complicating the tubercular disease, but nothing of the kind was found. It was a simple case of hydrocephalus. There was no infection of any kind as a result of the punctures.

DR. DAVIS.—The question of lumbar puncture must always be considered in the treatment of hydrocephalus. In this case, Dr. Keen preferred to drain the ventricles by the method which he has employed successfully in a number of cases.

Although drainage of the ventricles may for the time being give satisfactory results, we have no guarantee that the gain will be permanent. Cases are reported in which the cranium continued to enlarge after fluid had been removed.

As regards immediate danger of death, it seems to depend largely on the amount of fluid withdrawn, and hence those who perform this operation are solicitous that as little fluid as possible should escape. The effort is made to maintain the intracranial pressure, to dam up the fluid and secure a slow and aseptic emptying of the ventricles. Even under the most favorable circumstances, the results of this method of treatment are not encouraging.

DR. ROTCH.—In the cases we have operated upon at the Infants' Hospital the greatest care was taken, and some very good scientific and practical work was done in keeping a record of the amount of fluid pressure. The observations were made by Dr. John Dane, by means of a manometer attached to the drainage tube. The positive pressure was found to be 7 cm. and when the child cried it rose to 12 cm. The pressure was found to vary considerably. I have already reported the case.

INTESTINAL OBSTRUCTION THROUGH A LOOP FORMED BY MECKEL'S DIVERTICULUM WITH LIGAMENTOUS ATTACHMENT.

BY IRVING M. SNOW, M.D.,
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Intestinal obstruction is often a very obscure condition, being caused by many complex and interesting pathological changes. The following case seems worthy of record, as the stenosis of the bowel was the result of a curious congenital deformity. The patient was a boy of three years, in good health up to the present illness.

Nov. 9th.—He fell a short distance from some steps, hitting himself on the abdomen to the right of the navel. No bad effects followed this accident; in fact, there was complete relief in an hour.

Nov. 11th.—The child ate quite freely of grapes, and there is some reason to believe that this dietetic indiscretion, by producing intestinal irritation and increased peristalsis, was the exciting cause of the obstruction.

Nov. 12th.—The boy had a normal fecal passage, but commenced vomiting, and vomited persistently for four days. During this time in spite of laxatives and enemata, the bowels remained obstinately constipated until Nov. 17th. There was also agonizing, nearly continuous, abdominal pain, so intense as to require opiates on Nov. 14th, 15th and 16th. When the child was asked where it hurt him he pointed always to the navel. Nevertheless, there was no tenderness, induration or abdominal tumor to be felt; the belly was slightly distended, with some gurgling and rumbling on palpation. Temperature insignificant—0.5° to 1° above normal; indeed, the illness ran its entire course without fever.

On November 16th and 17th marked prostration developed from the pain, lack of nourishment and frequent vomiting. I first saw the child on November 17th, on the sixth day of its illness. He was a stout little boy, slightly narcotized, with slow, sighing respiration; pulse, 120; temperature, 99°; lungs, throat, heart and skin normal; abdomen tympanitic, somewhat distended; no hernia, no abdominal tenderness, doughiness nor induration; rectal examination negative.

Late in the afternoon the child vomited fecal matter. To remove a supposed fecal impaction a Nobel injection was given. The effect of the injection was to cause intense abdominal pain;

the child writhed with colic, vomited fecal matter again, and went into profound collapse; after several hours a small fecal stool containing three grape seeds was passed. The evacuation was followed by the expulsion of considerable wind, and by a subsidence of the abdominal distension. Careful palpation of the relaxed abdomen revealed no tenderness or abnormal swelling.

Nov. 18th.—Child apathetic, frequent sighing; occasional abdominal pain; some tympanitis; respirations, 12; pulse, 120; temperature, 99.5°.

Fig. 1. STRANGULATION BY MECKEL'S DIVERTICULUM, AFTER TREVES.

Nov. 19th and 20th.—There was now observed visible peristalsis which lasted until the child's death.

Nov. 22d.—Patient again commenced to vomit fecalent matter; a quart of olive oil was given by enema. A fecal stool containing grape seed, accompanied with flatus, was the result of this procedure; no fecal matter was vomited after the olive oil injection, which, in fact, seemed to relieve the colic, and to remove a lingering doubt as to the presence of a complete intestinal obstruction. Nevertheless, the patient failed rapidly, and died November 23d after an illness of twelve days. The cause of death was shown by the autopsy to be exhaustion and inanition from pain, vomiting and interruption of assimilation from an unrecognized abdominal obstruction.

Autopsy.—No peritonitis; appendix healthy. The symptoms were due to the snaring of a coil of ileum in a loop formed by an intestinal diverticulum projecting from the ileum and connected by a slender ligament to the mesentery close to the ileocecal valve. This glove finger-like diverticulum (Meckel's) was given off twelve inches from the ileocecal valve and measured two and a half inches in length.

The Meckel's diverticulum and ligament formed a loop

going one and a half times around the ensnared intestine. In the loop the diverticulum lay uppermost, forming the upper and about one-half the lower portion of the constricting band. The strangulated intestine was the portion of ileum between the diverticulum and the ileocecal valve; the neck of the coil of intestine was not tightly compressed, and could easily be slipped to and fro through the loop. Evidently the constriction did not completely close the lumen of the bowel, as no fecal matter was found in the small intestine, and above the incarceration we found some olive oil, forced through the ensnared gut by an enema given a day before the child died. Above the obstruction the ileum was slightly distended. No congestion or discoloration of the small intestine was observed.

This form of intestinal obstruction has been exhaustively described by Treves. In his classification of intestinal stenosis by bands and apertures he states that strangulation by the diverticulum and diverticular bands through loops and knots forms one-third of the total number. These are relatively rare in early childhood. The mechanism of the snaring of the bowel is very simple. Meckel's diverticulum is the persistence in extrauterine life of a fetal structure—the omphalomesenteric duct. In its most perfect state it exists as a tubular canal extending from the lower portion of the ileum to the umbilicus. More frequently than this canal a blind tube two to four inches long is present. Occasionally a fine ligament, the obliterated portion of the omphalomesenteric duct extends from the end of the diverticulum to the abdominal wall near the navel or breaking loose from this region contracts secondary adhesions generally to the mesentery.

Thus the diverticulum and ligament may form a loop floating free in the abdominal cavity. Exaggerated or irregular peristalsis may cause coils of intestine to pass through the arcade thus created, and by the tightening of the loop stenosis or complete intestinal strangulation occurs. In my case the bowel was not tightly compressed; fecal matter, grape seed and flatus passed through the constriction, and olive oil was forced above it. It must also be remembered that fecal vomiting is a frequent accident after an intestinal irrigation.

This seeming perviousness of the intestine caused a fatal error in diagnosis. The condition was not recognized as intestinal obstruction, and not being relieved by operative treatment, ended in death.

DISCUSSION.

DR. CAILLÉ.—The case reminds me very much of the cases of chronic intussusception, if I may call them so, in which the clinical symptoms are those of obstruction, and in which the obstruction is not absolutely complete; and I think in every instance there should be an exploratory laparotomy. It seems to me that if an exploratory laparotomy had been done in this case on the first occasion of fecal vomiting, there might have been an opportunity of saving the child.

DR. ADAMS.—I am in favor of early operation. I have so often seen hospital cases in which after delay the surgeon says, "I will operate and give the child the only chance it has," with the result that in five or six hours the child is dead. In a case that recently was under observation, the baby was taken ill Sunday afternoon, and Monday morning when I saw him he had vomited, but there had been no fecal vomiting. There was the characteristic strain and scream; no tumor could be made out, but he had an anxious expression. I advised the parents to have a surgeon in consultation. The father had unfortunately given in the morning three drops of a deodorized tincture of opium which had been in the house for some time, and the baby was completely narcotized. The baby was given injections of the normal saline solution, with apparently some relief. The abdomen became very much distended, however, and the next day the surgeon was called in. The anesthetic was given by me, and after the baby was under the influence of the anesthetic he had a profuse stool, consisting of fecal matter. The question then arose whether the obstruction had been relieved. The surgeon, with others present, thought, as the abdomen had not collapsed and no flatus had been passed, he had better operate. He operated and found an invaginated intestine of about four inches, but not adherent. That baby, between six and seven months of age, recovered. It is the only case in the District of Columbia of an infant that has recovered from the operation. So that has encouraged me to advise early operation. I think the trouble has always been in waiting and attempting to untangle, so to speak, the intestine either by the introduction of gases or fluids. Now we know the history of these cases. It certainly is plain enough, they go on from bad to worse, and very few of them spontaneously recover.

DR. CHRISTOPHER.—I would like to report briefly an intensely interesting case in a breast-fed, perfectly healthy baby four months old. It was sick in the morning, I saw the baby at twelve o'clock and it had then a temperature in the rectum which was normal, and some normal feces were passed. The most careful examination failed to reveal any trouble. Intussusception was the only thing that we could make out. At five o'clock the baby was seen with me by Dr. Henrotin with a view

to operation. At six o'clock the abdomen was opened. There was no vomiting at any time and it simply presented the appearance of a normal healthy child with great depression. Intussusception of an inch and a half of the small intestine into the colon was found. This was removed very readily and the baby made a complete recovery, nursing the mother half an hour after the invagination was reduced. When seven months old the same condition recurred, the diagnosis being made by the mother. Dr. Henrotin and I arrived at the house about ten minutes apart and agreed with the diagnosis of the mother. We attempted to reduce the invagination but failed. In about six hours the child's abdomen was opened and again an invagination was found. There was the same lack of symptoms. While the abdomen was open we attempted to reduce the intussusception by the injection of air, and I believe we could have put on pressure enough to have ruptured the intestine without any reduction. The intussusception was then reduced by hand, and advantage taken to remove the appendix. The patient made a complete recovery, and has gone a year and a half now without intussusception. So far as I know it is the only instance in which there has been a double intussusception in the same case with recovery of the patient.

DR. CARR.—This history emphasizes the importance of early operation in this class of cases where there are no symptoms that are considered diagnostic. In the past year I have had three cases in which there has not been a rectal tumor. Another thing, some of these cases have fecal movements through the intussusception. Many of these patients show symptoms that are hardly more severe than are seen in an ordinary colitis. The symptoms are very frequently due to obstruction that is not recognized. In one case the surgeon opened the abdomen under protest. He did not regard the symptoms as due to intussusception, but he found it. The English surgeons have recorded a number of cases like Dr. Snow's in which on making an incision they found a constriction of Meckel's diverticulum. The general symptomatology may be wanting, but it is a condition that calls for prompt surgical interference. It cannot be relieved by injections either of water or of air.

THE PRESIDENT.—One point in Dr. Snow's paper seems to have been overlooked: that is, the oil was found above the intussusception, showing that the oil injected got past the intussusceptum but did not reduce it.

DR. SNOW.—I spoke of early operation and stated that operation was delayed on account of the obscurity of the symptoms. Strangulation through a loop formed by Meckel's diverticulum is very rare and is difficult to diagnose. The only satisfactory treatise on the subject is that of Treves. In cases similar to the one I have described, early operation is indicated even if there is a doubt as to the diagnosis.

THREE CASES OF HEAD-NODDING AND HEAD-ROTATION IN RACHITIC INFANTS.

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The comparative rarity and interesting nature of that curious functional nervous affection of young children variously known as spasmus nutans, gyrospasm, rotary head spasm, head-nodding and head-jerking, and which is often associated with nystagmus, is regarded as a sufficient reason for presenting to your consideration 3 cases observed by me during the past ten years. It is a source of regret that these cases were not more thoroughly studied, especially the eye-symptoms, for neither at the time they were observed, nor when it was decided to present them to this Society, was I alive to the close and important relation which these symptoms bear to the symptomatology and etiology of the affection.

The infrequency of the disorder is shown by the statement of R. W. Raudnitz[1] that in the Budapest Children's Hospital, during the years 1890 and '91, and in 1894, but 14 cases occurred among 52,213 patients, while a very thorough search of the available literature has revealed only 78 instances of the affection,* distributed among various authors, as follows: M. H. Romberg,[2] 2; Eberth,[3] 2; E. Henoch,[4,5] 8; S. Mackenzie,[6] 2; S. Gee,[7] 3; Gorden Norrie,[8] 3; W. B. Hadden,[9] 21; Ed. Tordeus,[10] 1; Buzzard,[11] 1; A. Caillé,[12] 2; Eschoua-Fridmann,[13] 3; George Dickson,[14] 1; Wm. Hirsch,[15] 1; Fraenkel,[16] 1; R. W. Raudnitz,[1] 15; F. Peterson,[17] 5; A. Hand, Jr.,[18] 1; C. J. Aldrich,[19] 2; C. F. Judson,[20] 1; D. J. M. Miller, 3.

It is not my purpose to discuss at length the causes, clinical features, and pathology of this functional disorder. This has

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*Since the above was written 6 more cases have been reported: 2 by I. A. Abt (*Journ. Am. Med. Asso.* 1900. xxxiv., 269,) and 4 by Ausch (*Archiv. f. Kinderheilk.* 1900. xxxiii., 161.)

been so thoroughly done by Mills[21], Hadden, Raudnitz and Aldrich that little remains to be said. I propose merely to report 3 cases, and then to analyze, in as brief a manner as possible, the reported cases, with reference to the significance and comparative frequency of the principal symptoms and etiological factors.

Before going further it will be well to direct attention to the misleading character of the terms by which the disorder is generally known, particularly the term spasm, which by no means describes the movements, which are, as Aldrich has well pointed out, "monotonously regular, smooth and easy." The title, "head-nodding and head-rotation," suggested by this observer, being much more appropriate and descriptive.

CASE I.—Geo. M., colored, five months, breast fed, was first seen February 3, 1898. He had been subject to colic, vomiting and obstinate constipation since birth. The belly was protuberant and flaccid, the fontanelles large and the teeth absent. The ribs were slightly beaded and the epiphyses of the wrists and ankles enlarged. He weighed at birth eight pounds, at the time of his visit ten pounds, fourteen ounces. Under the use of massage, salt-baths, cod-liver oil and artificial food he improved somewhat, but was soon lost sight of. On May 27th (nine months old), he was brought again because of peculiar movements of the head and eyes, which had begun after an attack of measles some two months before. Because of this illness (measles) he had been weaned entirely and fed upon cow's milk and Eskay's food. The evidences of rickets had markedly increased. The bowels were still obstinately constipated and the tongue coated. The two lower central incisors were just emerging. The head movements were vertical, *i.e.*, up and down, occasionally somewhat lateral, with an inclination of the head to the right. The movements were quite rhythmical and without spasmodic character. They occurred about ninety times a minute, and ceased while feeding, and for a few seconds only, if the attention was suddenly directed to some object, or by snapping the fingers quickly. They were much less marked in the recumbent position, and ceased during sleep. The nystagmus, which had appeared about a week after the head movements, was much more rapid than the latter, and was chiefly vertical, occasionally diagonal, and was present in whatever direction the gaze was directed. The pupils were equal and reacted to light. The fundus was not examined.

The knee-jerks were normal. At a subsequent visit, one month later, it was noted that the head movements were less marked, and that the nystagmus was almost entirely vertical. Two months after this I saw the child again. It had much improved in health and the upper incisors were beginning to emerge. The head movements and nystagmus were only occasionally seen, the mother said only when excited. An examination made the following October failed to discover either head or eye movements, the mother had not noticed them for a month or more. They lasted altogether about four months.

CASE II.—D. H., white, female, twelve months old, breast-fed for three months, since on condensed milk. Was first seen at the Children's Hospital, in February, 1895. Alternating diarrhea and constipation had been present for many months. Very marked rachitic symptoms were noted: protuberant belly, wide fontanelles, no teeth, inability to maintain the erect position, enlarged epiphyses and well-defined rosary. For five months, *i.e.*, since the seventh month, the head movements had been noticed. They were lateral, or rotary, rapid, smooth and rhythmical, occurring fifty to sixty times a minute, and ceased when lying down, when asleep, and momentarily, when the attention was suddenly fixed. On attempting to arrest the movements by firmly grasping the head, a rapid, horizontal nystagmus of the left eye became apparent, which ceased when the head was released. The pupils were equal and reacted to light. Both knee-jerks were present. The fundus was not examined. At a subsequent visit I was told by the mother that the child had a peculiar way of holding the head to the right when looking at objects. Some months later I learned from the mother that the movements had entirely ceased, and that the child was much improved in health and could sit up alone. The attack lasted about seven months.

CASE III.—Infant of eight months, seen but once (in March, 1890), female. Fed upon condensed milk from birth. No teeth. Marked evidences of rickets: enlarged epiphyses, large fontanelles, protuberant abdomen and beaded ribs. The tongue was coated and the child subject to attacks of diarrhea. Mother brought the infant because of lateral movements of the head which had existed for two months, and which were only noticed in the erect position. There was no nystagmus, nor could a history of such be obtained. Further course unknown.

The treatment of all these cases consisted in regulation of the diet, salt baths, massage, sunshine, fresh-air and cod-liver oil.

As already mentioned, it is a source of regret that these cases were not examined by a trained ophthalmologist; but this does not detract from the faithful picture that they present of the curious syndrome, or affection, so graphically depicted in Hadden's classical papers. Of particular note is the association of all of them with well-marked rachitis, a connection which has been remarked by most observers. It was present in 9 of Hadden's 21 cases, and in 39 of the 78 cases above referred to, or in 50 per cent. It is probably the most potent, exciting or predisposing cause. Another recognized exciting influence also existed in my cases, namely: gastrointestinal disturbance, although its occurrence is only noted twelve times in the reported cases. The rôle played by acute illnesses in precipitating an attack, or inducing a relapse, was exhibited in my first case, which was immediately preceded by an attack of measles. In 9 other instances this exanthem preceded a first or second attack. This connection is of interest, because of the conjunctivitis accompanying the latter affection and its possible relation to the nystagmus. In 2 cases the symptoms were preceded by pertussis, in 1 by erysipelas, and in 1 a relapse was induced by pneumonia. Further study of the 78 cases elicits the following interesting facts: In 11 there was a personal history of convulsions, and in 12 a similar history in other members of the family; in 4 cases other children of the family, or the mother, had had chorea; 27 were males, and 27 females, the sex not being mentioned in 24 cases.

Falls, which Peterson and Hirsch consider the most important exciting cause, occurred in 22 cases (28.2 per cent.). As to the age of onset, in 2 the affection began respectively at the age of four (Gee) and six (Hadden) weeks, and in 1 at three years (Henoch); 5 began between the third and fifth month, 51 between the sixth and twelfth months, 16 between twelfth and eighteenth months, while 1 case occurred at nineteen and 1 at twenty months. The disorder, therefore, is most frequently encountered between the sixth and eighteenth months (67 cases), a period when the evolution of the teeth is most active, but it is very questionable whether dentition has anything more than a feeble, exciting or predisposing influence.

In 2 of my cases the head movements were horizontal, *i.e.*,

from side to side, while in the other they were distinctly vertical, or nodding, occasionally interrupted by lateral movements. Horizontal, lateral or shaking movements are by far the most common; they occurred in 49 of my series. Purely nodding movements I find in only 12 cases, instances of this character being usually associated with occasional lateral movements. In 18 cases the movements were combined rotary and nodding, while in 3 no head movements were noted. A peculiar manner of holding or cocking the head to one side on looking at objects, or throwing the head directly backward, is of frequent occurrence. Hadden observed it in half of his cases, and its presence was mentioned in 26 of my series. As observed by Aldrich, the direction of the head is usually towards the side of the monocular nystagmus. This peculiarity was present in my second case. According to Raudnitz, this phenomenon, as well as the head movements, are dependent upon the direction of the gaze (Blickrichtung), as they both cease when the eyes (one eye if the nystagmus is monocular), are bandaged.

Study of the reported cases shows that the nystagmus may present itself in every conceivable form. In my first case, where the movements were nodding, and, occasionally, lateral, the nystagmus was partly vertical and partly diagonal, the former preponderating. In the second case the usual correspondence of the movements of the eyes and head was present, and, as has often been observed, of the nystagmus alone, when the head is firmly held. The direction of the movements of the head and eyes usually correspond. In 46 cases of rotary head movements, the nystagmus was horizontal in 36, mixed in 5 and in 5 its character was not mentioned. Simple nodding movements are usually associated with vertical nystagmus, but not always; in 7 instances of this variety the eye movements were absolutely vertical in only 3. Other interesting points in connection with the ocular symptoms brought out by a study of 78 cases are as follows: The nystagmus was binocular in 43, monocular in 22, and absent or not mentioned in 13. In 49 cases in which the character was noted, the nystagmus was horizontal in 36, vertical in 3, lateral and rotary in 5, lateral and sometimes rotary, sometimes vertical, in 6, vertical in one eye and horizontal in the other in 2, and in 3 occurred without head movements. In 5 cases there was strabismus without nystagmus; in 2 of these it preceded the other symptoms, and in 1 was the

only ocular manifestation present. The upper eyelids took part in the nystagmus in 4 instances; the later was vertical in 1 of these, and in 3 vertical, diagonal and horizontal. The eyes were ophthalmoscopically examined in 22 cases and found normal in 21; in the other (Hadden's) there was a slight crescent atrophy around the disc.

Critical examination of the recorded cases of the affection forces the conclusion that it presents a sharply defined clinical picture which ought not to be confounded with many other conditions with which observers in the past and present have associated it, and with which it has nothing in common. Such conditions are that peculiar form of epilepsy, known as tic salaam, the imperative and automatic movements of feeble-minded children, habit spasm, head-banging and other choreic affections, juvenile or congenital nystagmus and some forms of petit mal. The favorable and brief course, usually from a few weeks to a few months, the invariable recovery, the absence, in the vast majority of cases, of anything approaching epileptic paroxysms (I exclude the losses of consciousness noted in a few instances by Hadden as incidental occurrences, and not as essential parts of the affection), the lack of signs of mental deterioration (many of Hadden's patients seemed to be particularly bright), and the invariably normal condition of the optic discs, serve to separate these cases from more serious organic affections.

As to the pathology of the disorder, I have nothing new to suggest. Two of Raudnitz's cases came to autopsy. In neither was any change found, macro- or microscopically, in brain, medulla, optic nerves or muscles. Hadden believes that the movements are due to an "instability of the motor centers above the nuclei, in the spinal cord and fourth ventricle, presumably the cerebral cortex." The young child's purposive movements are not yet firmly fixed, and are easily disturbed or disarranged by some functional disturbance. Aldrich would refer the condition to cortical exhaustion, or to disease or defects in the medullation of the conducting fibers, brought about by rachitis or other nutritional disturbances, the unstable nervous system of the infant taking on such erethistic sensibility that any slight excitation may set up an irritative discharge of nerve force that finds expression in the movements of the head and eyes.

In 1889 Caillé reported to this Society two cases of nystag-

mus with head movements in rachitic babes, in whom the movements ceased on bandaging the eyes. He suggested as an explanation that the muscular spasms were either compensatory or reflex from the irritation caused by light to those structures concerned in carrying the impression. It is curious that no other observer should have been impressed by the importance of this observation. Hadden, it is true, refers to it as probably correct, but says he tried it in only one case, and Aldrich quotes Mills as making the same remark, but finds little authority for the statement. Raudnitz, by means of a series of painstaking tests, carried on through a long period on the same patients, claims to have proved that it is the direction of the gaze, or line of sight (Blickrichtung) that causes and governs the movements, so that they cease when the eyes (one eye if the nystagmus is monocular) are bandaged, and this even occurs when the nystagmus is not present. This latter phenomenon he believes exists in all cases—without exception, at the height of the disease. That it has so often escaped detection is due to the fact that the cases were not observed during their whole course (often only once), or was not present at the time of examination, or was not elicited by well-directed tests. All observers have noted that this symptom may be present at one time and absent at another, or only discovered by placing the eyes in certain positions.

The eye and head movements, therefore, according to Raudnitz, depend upon the line of sight—a reflex spasm brought about by the attempt of fixation, mostly developed in weakly children living in dark and gloomy apartments. Almost all of his patients lived in dwellings of this character and occurred in the winter months. The crib or cot in which the child lay was so placed in most cases that the window or a bright light or reflection was constantly looked at in an unnatural manner. The condition, in short, he makes analogous to miners' nystagmus. From this point of view rickets, intestinal disorders and acute illnesses would only act in a predisposing or exciting manner by producing local (eye) and general muscular weakness. This explanation will certainly not apply to all cases. I visited the houses of two of my patients several times and did not notice that the rooms were particularly dark—indeed, one of them was very light and had been occupied many months before the onset of the movements. It is evident that the nature of the

affection is still very obscure, and cannot be explained by any one theory; but its functional nature is conceded by all and it is probably due to an exhaustion or irritation, induced by rickets and other causes, of the ganglion cells innervating the muscles of the eye and head. In the treatment the administration of bromids, so generally recommended, is not so necessary as attention to diet, hygiene, etc., and the administration of drugs directed to the improvement of the general health and the relief of any constitutional vice or temporary ailment from which the infant may be suffering.

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DISCUSSION.

THE PRESIDENT.—In my own clinic, one of my assistants, during the past year, collected a number of cases of rotary spasm associated with nystagmus. He established the fact that, as a rule, nystagmus is more marked in one eye than in the other. We have also found if you fix the vision of the

child you may bring out very slight nystagmus that otherwise might not be observed. Another assistant made the observation in my clinic that many of these children live in very good apartments and are taken out almost daily. The theory that there is eye strain in these cases is not carried out by investigation. We have now a very bright baby with rotary spasm, apparently perfectly well, with no gastrointestinal disturbance. The only abnormal condition present is rachitis. There is very marked craniotabes. This subject of rotary spasm and nystagmus opens a very wide field for the neurologist.

DR. MILLER.—Mr. President, I will only say that the few cases I have observed have not been conclusive. Two of my cases did not certainly live in dark rooms, and I only refer to this as suggestive and not by any means as a solution of the etiological problem. But all my cases presented marked evidence of rickets. Rickets is found so often that it probably is present in slight degree in almost, if not in all cases, and may not be recognized because it is so slight. I believe the symptoms are due not only to eye strain, as they probably are in some cases, but also to exhaustion by rickets, or other conditions, of the nerve centers that preside over the eye and head movements.

NASOPHARYNGEAL DISEASE IN PEDIATRIC PRACTICE: A CLINICAL STUDY.

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Nasopharyngeal disease in pediatric practice may be viewed from one of two standpoints—the specialist's or the general practitioner's. The former is apt to see the cases late, when serious secondary troubles may have developed; the family physician, on the other hand, is more likely to be consulted at an early date. It is, therefore, important that the possible evil influences, direct or indirect, exerted by nasopharyngeal troubles generally, and adenoids in particular, should be kept in mind—otherwise the treatment will be symptomatic and palliative, rather than radical and curative.

The symptoms vary with the individual. In one the brain receives the brunt of the attack, in another the chest, in others circulatory or digestive disturbances are manifested, and so on. In some the relationship is evident, in others a careful study only will clear up the case. Much may be accomplished in the way of prophylaxis by a correct and early diagnosis.

The following, taken from Jacobi's masterly, instructive and scholarly article, "Some Preventives," is suggestive and will serve as our text: "Nasal catarrh, with its hyperemia and soreness of the mucous membranes, predisposes and causes chronic hypertrophy, adenoid growths, tumefaction of submental and submaxillary lymph bodies, invasion of diphtheria and tuberculosis, and occasionally meningitis." *

It is not to be inferred from the above that adenoids are the result of repeated attacks of nasal catarrh in all cases. In numerous instances, particularly when occurring in families free from syphilis or tuberculosis, the lymphoid hypertrophies must be regarded as the local manifestation of a constitutional dyscrasia, to which the term lymphatism has been applied.

In quite a number the trouble is congenital or shows itself within the first few months after birth. As lymphoid hypertrophies in the upper and middle pharynx are frequent in chil-

* *Philadelphia Medical Journal*, Dec. 10, 17, 24, 1898.

dren, it seems but logical to conclude that the enlargement of the pharyngeal tonsil in many cases is primary, and the catarrhal condition of the nasopharynx, particularly when attended by a semipurulent discharge, secondary — an effect and not the original cause. Our work will be facilitated and the ground cleared for subsequent discussion in detail, if at this point we refer to the functions of the nose and indicate thereby the anatomical relations of the nasopharynx.

The main functions of the nose are:

(*a*) Respiratory,
(*b*) Olfactory,
(*c*) To give resonance to the voice,
(*d*) And to act as a regulator of the aëration of the middle-ear,

and, we may add, of the accessory air chambers or sinuses in the frontal, maxillary, ethmoidal and sphenoidal bones.

Two conditions, patency of the nose and throat, and a healthy mucous membrane, are essential to the proper performance to the work. Disease, with a greater or less degree of stenosis, shows its evil effects in many ways, to be discussed later on.

The nasopharynx serves as a common area of air communication between five openings. The Eustachian tubes, one on either side, posterior to the nasal choanæ, ventilate the middle-ear. The acuteness of hearing depends upon the patency of the openings with free nasal respiration. The posterior nares also open into this space. They act as the normal channels for the passage of air through the nares to the lungs. Unobstructed nasal breathing is essential to the proper ventilation of the accessory sinuses of the frontal, superior maxillary, ethmoidal and sphenoidal bones. Finally, at the lower portion, communication is established with the oropharynx.

As a pathological entity encroaching upon or invading this space, we frequently meet with a hypertrophied condition of the lymphoid structures (Waldeyer's Tonsillar Ring). The symptoms are local and general. Some are caused by pressure, others are inflammatory in character, and many are the result of anatomical changes more or less permanent.

A discussion of the topic may appear trite to the specialist. It cannot be told too often to the general practitioner. Specialists, as a rule, do not see the cases early; the general practi-

tioner, on the contrary, is frequently consulted at a time when a recognition of the trouble enables him to ward off many outward evil effects by proper local treatment, operative or otherwise.

Though nasopharyngeal troubles are very common, in general practice, unfortunately, they are frequently overlooked, treated lightly, or dismissed with a few general directions. This is a serious error. Parents must not be led to believe that the child will outgrow the disorder, or that the symptoms will disappear about the time of puberty.

Advice of this sort, with neglect of appropriate measures, is certain to be detrimental to the mental and physical welfare of the patient. The popular belief, that operations upon the tonsils, etc., may be followed by defects in speech or imperfect development of the genitals, must be combated. Parents often refuse operative interference, until assured that no evil results will follow in this respect.

The family physician does well to remember that his duties are not confined to the treatment of an individual case or disease. Children under his care ought to be regarded as his wards from a medical standpoint. With a history of recurring attacks of nasal catarrh or mouth breathing, the dangers should be made clear to the parents. Unnecessary delay or procrastination must be avoided.

The attendant should bear in mind that the effects are not altogether local. Disturbances, cerebral and nervous, due to the obstructed blood and lymph circulation at the vault of the pharynx and base of the brain, are frequent. Deformities of the chest, bronchial and pulmonary inflammations, are common, as are recurring attacks of catarrhal croup.

The general circulation is interfered with, respiration, digestion, etc., disturbed, and dyspnea may be present. The poorly developed muscles, with lowered vitality in general, lead to chronic invalidism or render such patients an easy prey to acute disease.

In addition, enlarged lymph nodes at the angle of the jaw, repeated attacks of nose-bleed, acute and chronic bronchitis with emphysema and asthma, bronchopneumonia, large bronchial and mediastinal lymph nodes, are frequently secondary to a morbid state in the nasopharynx. The paroxysmal nocturnal cough, quite common in children, distressing and alarming

in character, disappears when the nose and throat are treated. The special senses, taste and smell, are more or less impaired in older children. The voice is altered and assumes a nasal character. Inability to pronounce the letters *m* and *n* and in some cases stuttering exist.

Diseases of the eye may be reflex or arise from a direct extension of the process in the nose. Most commonly there is direct extension. Deformities in the nasal passages, acute or chronic catarrh, and adenoids give rise to affections of the lachrymal sac and conjunctiva.

Pupillary changes, photophobia, disturbed accommodation, strabismus, blepharospasm, etc., are at times of reflex origin. A normal mucous membrane is the best safeguard against the onset of a number of infectious microorganisms. The invasion of diphtheria, tuberculosis, and now and then meningitis, is favored by an abnormal condition of the nasal and pharyngeal mucous membrane. The best preventive, therefore, is to keep the mucous membrane in a healthy state. The eloquent appeals in favor of a routine nasopharyngeal toilet have aided somewhat in popularizing the method. In the tenement districts, where most necessary, the precautions are imperfectly employed or wholly neglected. In this connection it may be stated that, when a child with adenoids and associated nasal catarrh contracts diphtheria, an extensive surface is apt to be involved. The type will be severe, the progress correspondingly grave. On the other hand, children who "take cold" easily, who present but few evidences of lymphoid hypertrophy up to this time, often develop decided symptoms of obstructed nasal breathing after an attack of diphtheria, scarlet fever, or measles —at times, in spite of carefully conducted nasal toilet during the course of disease.

Small painless lymph nodes at the angle of the jaw, about the size of an almond, are common. Though frequently mistaken for tonsils, this is an error. They are due to infection from the nasopharynx, and point to the presence of adenoids or a moderate degree of nasal catarrh. If an exacerbation of the latter takes place or an infectious disease is superadded, the nodes begin to swell and become more or less painful. Under appropriate treatment with nasal injections and cold applications externally, the process subsides and resolution takes place. In other instances suppuration occurs, either nod-

ular, perinodular, or both. Now and then the capsule becomes thickened and the process remains quiescent; sometimes caseation takes place or calcareous or fibroid degeneration occurs. Other chains of lymph nodes may be involved, the process extending downward to the bronchial lymph nodes. The chief danger, however, lies in the tendency to become tubercular. In the latter case, the process may remain local, infect other lymph nodes and tissues in the vicinity, or general tuberculosis may result eventually.

Surgeons, recognizing the danger, advocate and practice the removal of enlarged or tubercular cervical lymph nodes. Yet adenoids and large tonsils have been allowed to remain, to serve as a nidus for subsequent infection. They, as well as the external lymph nodes, ought to receive surgical treatment.

A large proportion of ear troubles, from 60 to 75 per cent. according to different authorities, are secondary to diseases of the nose and throat.

Adenoids, in particular, constitute an all-important etiologic factor. In nearly every case, ear disease is certain to follow and no time should be lost in advocating their removal as a prophylactic measure. Clifford Allbutt says the very worst degrees of depressed ear-drums are found in those affected with large growths. Deafness, deafmutism, and ear disorders in general are benefited at times by local treatment of the throat. In the course of the exanthemata and other infectious diseases, suppurative otitis with perforation is very apt to develop whenever a prior inflammatory irritation or congestion of the nasopharynx is present. The danger is increased if the pharyngeal or faucial tonsils are hypertrophied. Otitic troubles arise in several ways.

The Eustachian tube may be occluded with mucus, the pressure of adenoids against the orifice may cause its obstruction, and thus interfere with the proper ventilation of the middle-ear, or the catarrhal inflammation may extend through the tube and involve the delicate structure of the ear.

Trousseau, years ago, and others since then, have called attention to recurring attacks of erysipelas of the face in chronic aural or nasal catarrh with erosions of the skin. New outbreaks are avoided when, as a prophylactic measure, the primary condition of the ear, nose or throat is relieved. A few cases of this kind have come under our observation at the

Vanderbilt Clinic. The same is true of dermatitis and eczema under analogous conditions.

A word as to general diseases accompanied by local throat or nasal symptoms. In tuberculosis, syphilis and rheumatism,* and in the acute infectious diseases, the general characteristics are such that the nature of the local condition does not remain in doubt for any length of time. Now and then some difficulty may be met with in diagnosis.

Anatomists have clearly demonstrated the direct lymphatic communication between the vessels in the nasopharyngeal mucous membrane and those at the base of the brain. Bacteriologists have reported the presence of microorganisms in the nose and throat similar to those found in many cases of meningitis. Clinical observations show that the different varieties of meningitis are most commonly observed between the ages of three and five years, at a time when nasopharyngeal troubles are very common. The intimate lymphatic connection referred to, and the identity of the microorganisms in the nasopharynx and those found in a large number of cases of meningitis, tend to explain the etiology and many heretofore obscure inflammations of the brain and meninges.

A general infection by way of the blood must be distinguished from a local infection arising from some region in the neighborhood of the skull. A frequent mode (beside the one referred to above) is through the Eustachian tube to the middle-ear and thence to the cranial cavity. As a result, thrombosis, sinus-pyemia, inflammation of the meninges and brain, with or without abscess, are not infrequent.

Growth in general is more or less interfered with in many instances. Ewing, in an excellent article directing attention to the work done abroad, presented additional facts showing the diminished power of resistance, with the liability of sudden paralysis of the heart, in many of these patients.

Furthermore, a number of cases of sudden death during anesthesia for the removal of adenoids have been collected by Hinkel.

Deformities of the thorax, due to adenoids, are met with, though it should not be forgotten that other factors are usually associated. The worst cases occur in rachitic subjects, particu-

*Since the above was written, Dr. Packard, in the "Wesley M. Carpenter Lecture," discusses in an able manner "Infection through the Tonsils," especially in connection with acute articular rheumatism.

larly when bronchitis and pulmonary inflammation have been of frequent occurrence. The deformities vary in degree from the flat chest of the milder to the "barrel-shaped" and "pigeon breast" of the advanced type.

In seeking an explanation, it may be interesting to refer to the effects of nasal obstruction upon respiration, and to note the difference in the physical character of the air when it reaches the lungs in a normal manner through the nares, or abnormally by the way of the oropharynx. Inspired through the nose, the air is warmed, filtered, and moistened; in addition, further modifications occur from an interchange of gases between the blood and the atmospheric air.

When breathing is carried on through the mouth these changes do not occur, and the air not being filtered, warmed, or moistened, acts as an irritant. Consequently the delicate structures of the larynx, bronchi, etc., (rendered more susceptible because of the chronic catarrhal inflammation of the nose and throat, readily become inflamed. As the distal portion of the lungs do not expand fully under such conditions, the external atmospheric pressure being greater, the chest wall sinks in and deformities result. The degree varies according to age, the condition of the bony structure of the chest wall, the development of the muscular tissues, the presence of bronchitis and the amount of existing pulmonary collapse or deficient expansion. We are all perfectly familiar with the difficulty and discomfort experienced in breathing when afflicted with a cold in the head. The respiration becomes labored, and the lungs expand imperfectly for the time being.

In the case of the infant or child, the condition is more or less permanent, depending upon the degree of stenosis and the presence of acute or chronic catarrh. The breathing is superficial and the effects are more severe and lasting. Let any one attempt the simple experiment of breathing through the mouth for a short time, he will quickly realize the discomfort and fatigue, the dyspnea, sense of imperfect expansion, and the feeling of weight upon the chest.

In mild cases, in the young, the lungs expanding imperfectly, allow the thoracic walls to fall in, causing a shortening of the anteroposterior diameter. The chest becomes thin and flattened, the intercostal spaces are depressed, and the infra- and supraclavicular regions retracted. The Funnel Breast (Trichter-

brust), characterized by a funnel-shaped depression at the lower portion of the sternum, certainly, in some cases, is secondary to the nasal obstruction. It has been my good fortune to see a few in the process of development.

My experience accords with Osler, who says: "During inspiration, the lower sternum was forcibly retracted, so much so that at the height the depression corresponded to a well-marked Trichterbrust. While in repose the lower sternal region was distinctly excavated." A similar state of affairs was observed in an infant with a syphilitic affection of the nasal mucous membrane. The deformity disappeared as the nasal symptoms improved under antisyphilitic treatment.

In marked cases associated with rickets, the chicken or pigeon breast is observed. The sternum is prominent, particularly at the junction of the first and second portion, the ribs project anteriorly, while laterally, above the diaphragmatic or rachitic groove, the chest is depressed, giving a triangular shape to the thorax. In advanced cases, the chest is almost fiddle-shaped. In a well-marked instance in a child eighteen months old presented at one of our classes during the "Practical Course," it was surprising how quickly the deformity was remedied, when the patency of the nasopharynx was restored. The "Barrel Chest" is not infrequent, and occurs in those who are afflicted with chronic bronchitis, emphysema, and asthma. The neck is short, and round shoulders with or without scoliosis may be present.

In the absence of other causes, Coolidge believes that some of the atypical orthopedic deformities may result from a lowering of the general nervous vitality, frequently seen in patients with adenoids. Bilhaut found voluminous adenoids in many cases of scoliosis, removal of which at an early date brought about cure. Whatever the relation may be, it is important to secure pulmonary expansion in such cases, as the cure or improvement of the scoliosis is facilitated by furthering the development of the muscles and establishing good nasal respiration.

A practical point in hastening the cure of empyema may be incidentally referred to in this connection.

In a few cases of empyema in mouth breathers, curetting of the nasopharynx, by favoring pulmonary expansion through improvement in the breathing, caused the obliteration of a small cavity or sinus, thereby avoiding a secondary operation upon

the costal walls. In the same way, the associated lateral curvature rapidly disappeared when nasal respiration was established.

Snuffling in infants with retracted root of nose is of such evident import that even the tyro in medicine gives a correct interpretation. The nasal deformity should not be confounded with a similar state in cretinism and some forms of idiocy. The change in the appearance of the face, due to long-existing mouth-breathing, is characteristic, and admits of a ready explanation. The dropping of the lower jaw, due to a functional loss of tone in the muscles, adds to the length of the face, the latter appearing longer because of the deficient development of the superior maxilla.

The proper ventilation of the accessory sinuses or air chambers is interfered with by the nasopharyngeal obstruction. As a result, the blood supply is modified, normal growth of the bones does not occur, and expansion is retarded. The anemia and malnutrition, in consequence of the accompanying digestive and circulatory disturbances, leave their impress upon the face and give the drawn appearance to the eyes and mouth. The facial muscles are poorly developed, and the pinched nose or distended alæ add to the deformity. These changes, taken in connection with the mental state, give rise to the characteristic physiognomy.

A high-arched palate, with narrowing of the transverse measurements of the jaw, presenting a pointed appearance in front, with resulting contraction of the alveolar process, crowding and even rotation of the teeth on their axis, is frequent. The absence of the support of the tongue and increased atmospheric pressure upon the roof of the mouth, in consequence of the buccal breathing, explain the deformity. The gothic-shaped palate in turn crowds the septum, causing a deflection, and thus adding another factor to aggravate the inconvenience of the original trouble.

The teeth show a tendency to early decay, particularly the molars. In some cases, stomatitis and gingivitis occurs, persisting until the growths are removed. The breath is more or less offensive, the odor being caused partly by the bad teeth and partly by decomposed secretions, etc. Imperfect mastication, the rapid bolting of food, and the general anemia keep up and intensify the dyspeptic symptoms.

In younger children, particularly under a year, after exhaust-

ing disease with pronounced muscular weakness and relaxation, there is an additional danger, due to the tendency to falling back of the tongue and possible asphyxiation in consequence—particularly if the patient is allowed to sleep upon the back. Such cases must be carefully watched, and must be kept lying on the side. Strychnin and good diet soon restore the muscular tone.

Older persons frequently complain of shortness of breath. Talking, going up stairs, or rapid walking produces dyspnea and palpitation. A careful examination shows that the symptoms are due to the nasal trouble, and not to heart disease.

Nasopharyngeal obstructions induce abnormal breathing, anemia, disturbed sleep and a variety of nervous manifestations. The disposition is altered, the children become fretful or sullen, the memory is defective, and, apart from the impairment of hearing, such patients are inattentive, backward and dull.* In cases in which the growths have existed for a long time, the process may cause anatomical changes in the meninges and brain with resulting idiocy.

Headaches, often of a low grade, limited to the forehead and temple, may be accounted for by the retention of morbid products and obstructed circulation. Attacks of night terrors, walking in sleep, morbid dreams, melancholia and other evidences of disturbed cerebral functions may occur.

The mental and nervous phenomena are of extreme interest and importance. The question has been studied by Wells in an able and exhaustive article (*American Journal of Medical Science*, December, 1898), from which the following is quoted:

"Since we are dealing especially with psychopathic phenomena, how, we may inquire, can an obstructive lesion of the nose interfere with the cerebral functions? Briefly, by (*a*) alteration and impoverishment of the general, and secondarily of the cerebral, circulation, from the over-charging of the blood with CO_2 and the diminished supply of O, which are the necessary results of deficient aëration; (*b*) interference with the blood-supply of the brain by the lesion of the nose; (*c*) hindrance to the outflow of lymph from the brain. It has been shown that the subdural and subarachnoid lymph spaces are in direct connection with the lymph vessels of nasal mucous membrane. Guye held that aprosexia was owing to the interference with the lymph circulation, by reason of which the products of cere-

* Ribot holds that *acts of attention* are accompanied by a temporary suspension of the respiratory rhythm. The air hunger, depending upon the presence of adenoids, therefore of necessity interferes with the psycho-physiology of the act of attention. In this way he would explain the mental state of such children.

bral tissue metabolism are accumulated in the brain, producing brain fatigue or the so-called 'retention-exhaustion.' (*d*) It is barely possible that there may be some direct oxidation by the central nervous system, by means of the olfactory bulb (as in some animals) which function, if it exists in man, would be prevented by obstructive lesions of the nose."

Exceptionally a pure reflex case may present itself. In the vast majority, other causes exist, the removal of which yield brilliant results. In view of the marked improvement and entire disappearance of local facial spasms at times, following the relief of the nasopharyngeal disease, some relation of cause and effect must be admitted. The deleterious effects of the nasal disorder upon the blood and lymph circulation in the brain, and the accompanying anatomical changes, are responsible, in a measure, for the various neuro- and psychopathic manifestations occurring in *neurotic* subjects.

Jacobi, in an article published in 1886, directed attention to "partial, and sometimes general, chorea, minor from nasopharyngeal reflex." During the past ten years, the writer has seen quite a number of cases (at the Vanderbilt Clinic) improve under local treatment directed to the nasopharynx, arsenic being given at the same time, though arsenic and tonics alone failed to make much impression.

Nasal obstructions (inflammatory or otherwise) no doubt act as factors in the production of asthma, in a number of cases —not, however, as the sole agents. A neurosis which remains active after the nasal trouble is relieved is generally found.

In the discussion of this part of our subject, three conditions must be considered: First, predisposition, varying in degree in different individuals; second, some abnormality or undue excitability of the mucous membrane in some portion of the air passages; and, finally, a distinct irritant, which in consequence of individual idiosyncrasy, is reflected to, and again from, the respiratory center. The greater the predisposition, the less the exciting cause needs to be. The truth of this was frequently exemplified in our experience at the Clinic. In numerous cases, the tendency to bronchitis was relieved by freeing the nasopharynx. Yet the attacks of asthma would occur, sometimes less frequently; in other instances, no benefit resulted; now and then a cure was noted, probably in cases in which the predisposition was slight.

For a moment, attention will be directed to a brief study of the cases in which enuresis is observed. In some the incontinence is nocturnal, in a larger number it is both nocturnal and diurnal. A neurotic condition, with anemia and flabby muscles generally, is frequently found to be associated with the urinary difficulty. Increased thirst and polyuria add to the distress. As to an explanation, a plausible solution is offered in the mental state incidental to mouth-breathing. Apathy and listlessness, with disturbed intelligence and deficient innervation in general, are present—*conditions*, manifestly the result of the obstructed circulation in the blood and lymphatic vessels at the base of the brain and vault of the pharynx.

The higher inhibitory centers, for reasons given, do not act in a normal manner; the bladder reflex, consequently, is not respected, and incontinence follows. Furthermore, the frequent indulgence in water, to relieve the thirst caused by the parched condition of the lips and tongue, produces increased flow of urine, another factor in the etiology. Drugs are of very little service under such circumstances; to cure these patients, the pathological state in the nasopharynx must be removed.

An attempt has been made to present the more important features. Much might be added. The instructions in the following, taken from the paper of Jacobi, are to the point. If carefully followed many evils may be avoided and a great deal accomplished in the way of prophylaxis.

Jacobi writes:

"I have always made it a rule to keep all the integuments clean. At least once a day a physiologic solution of salt water is poured through the nares of every infant or child over whom I have control. Big adenoids should be removed, large tonsils resected. There is more danger in a dirty nose than in an unwashed face. Only do not be satisfied with merely ordering it. I have met many a 'trained' nurse who did not know how to inject or to irrigate a nose. A mother or a child's nurse should be instructed by you personally how to do it. Here, as everywhere, when two do the same thing, it is by no means the same. There are many cases of nasal diphtheria, such as are most likely to resist the influence of antitoxin, which are still spared a fatal termination by persistent and correct irrigation of the nares and nasopharynx.

"Pure air and sunlight are indispensable to health. The air should enter the lungs by way of the nasal passages; 'And breathed into his nostrils the breath of life,' we find recorded in Genesis.

"There is more than a grain of truth in the aphorism, 'Shut your mouth and save your life,' found on the title-page of Captain Catlin's celebrated pamphlet on mouth-breathing."

The homely, forcibly expressed dictum of Catlin must not be lost sight of. Give the little patients free nasal respiration, and give it to them early—the earlier the better.

Preventive medicine has done much to alleviate human suffering. Efforts in this direction have already borne fruit, and as a knowledge of etiology increases, advance in prophylaxis will keep pace.

These assertions are particularly applicable to our subject. To sum up, we may add:

1. The removal of the lymphoid hypertrophies in the naso- and oropharynx, with the cure of the associated nasopharyngeal catarrh, will restore the patency and permeability of the nose. If done early, many local pathological changes may be avoided.

2. The general health will be more or less improved.

3. The mental faculties and general intelligence will be improved.

4. Defects in speech and in hearing due to nasal troubles will disappear.

5. Deafmutism may be relieved.

6. The functions of taste and smell will be restored.

7. Reflex neurosis of various kinds will be modified or cured.

8. Nasal and supposed pulmonary hemorrhages will disappear.

9. Thoracic deformities will be relieved or cured.

10. The tendency to acute rhinitis, pharyngitis, laryngitis, bronchitis and pneumonia becomes less and less with the restoration of normal respiration.

11. The dangers attending the presence of enlarged cervical lymph nodes will be avoided.

12. The invasion of various infectious diseases is less likely when the nasal mucous membrane is in a healthy state.

13. The danger of meningeal infection from the nasopharynx will be lessened.

14. Ear complications in general, and particularly those incidental to the infectious diseases, will be avoided or rendered less dangerous.

PERFORATION OF A TUBERCULOUS BRONCHIAL LYMPH NODE INTO THE TRACHEA. SUDDEN DEATH.

BY A. CAILLÉ, M D.,

New York.

This specimen was taken from the body of a girl, four years old, who died suddenly in the Babies' Ward of the Post-Graduate Hospital. She was admitted by the house physician with the diagnosis, "bronchitis." There was no elevation of temperature, no pain, no dyspnea, and the heart and kidneys were free. A few râles could be heard on auscultation over the sternum. On the morning following her admission, she was playing with other children in the ward when she suddenly began to complain of pain in the neck, and almost immediately became cyanotic and asphyctic.

As a large caliber O'Dwyer tube did not relieve her dyspnea a low tracheotomy was performed also, without giving relief. The obstruction was evidently in the lungs and she died in a few minutes.

At the autopsy both bronchi were found plugged with a cheesy material which came from an abscess cavity situated above the bifurcation of the trachea, and which had perforated and ruptured into the trachea. As long as the child was under our observation there were no symptoms pointing to such a condition and the cheesy gland was in an unfavorable position for surgical interference.

At the February, 1900, meeting of the New York Pathological Society two similar cases were reported.

DISCUSSION.

DR. ROTCH.—I should like to ask Dr. Caillé what was believed to be the matter with the child before this was discovered.

DR. CAILLÉ.—The child was sent in by the family doctor with a diagnosis of bronchitis, and the house physician reported finding a few râles under the sternum. The child had no fever, hardly any cough, and was so playful and apparently in such good spirits that it was not put to bed at all.

DR. ROTCH.—It was supposed to be a tuberculous lymph node?

DR. CAILLÉ.—Yes; it was a tuberculous lymph node.

DR. ROTCH.—It is one of the most interesting cases. It happens I have been looking into the literature and asking the physicians their experience with tubercular bronchial nodes. They are a very important class of cases, and the question is, how are we going to detect them? I have been absolutely unable to find any symptomatology. Yesterday when Dr. Caillé read his paper on the tuberculous disease of the peritoneum we were forced to acknowledge how many children are tubercular whom we did not know were tubercular. It is a very serious matter, in a child with bronchitis, to have it suddenly die when it appears it should get well. But I am absolutely unable to find any symptomatology by which we can be aided in these cases. The bronchial nodes I suppose are the most frequent nidus of infection, and still those cases are more difficult to diagnosticate than the mesenteric, and, of course, more difficult than the cervical nodes. They often become encapsulated and we hear nothing further of them. But they should make us most guarded in our prognosis, whenever a child with enlarged lymph nodes has a cough. The specimen is a most beautiful one.

DR. CAILLÉ.—If there are any symptoms that will direct our attention to the condition it must be pain and dyspnea.

DR. ROTCH.—I would also suggest pain and edema and a certain amount of cyanosis of the face in addition to the dyspnea, but these symptoms often are not present.

DR. CAILLÉ.—That was the only tuberculous focus we could find in the child.

DR. FREEMAN.—I would like to mention the production of some cases of postpharyngeal abscesses as an analogous condition due to suppuration of tuberculous lymph nodes. Three cases of abscesses in the respiratory tract due to the breaking down of tuberculous lymph nodes have been seen at the New York Foundling Asylum in the past few years. One of these was mentioned by Dr. Caillé; it was low down at the bifurcation of the trachea; it broke into the trachea and the child suffocated. There have been two other cases in which the tuberculous lymph node was higher up and a postpharyngeal abscess was formed; the two children died from some other trouble and at the autopsy these cheesy nodes were found at the seat of the abscesses.

DR. ROTCH.—We should be careful in our prognosis in retropharyngeal abscess. The abscess should be opened, but the case should be watched closely as there may be suppuration in some of the deep seated glands and a grave condition may be present.

DR. DORNING.—Unless there is a large mass of the bronchial

lymph node, I do not believe there will be any symptoms at all. I would like to ask if any member of the Society has been assisted in diagnosis by a procedure that I have seen mentioned, and which I have tried? When listening over the sternum, with the head in its natural position, nothing abnormal may be heard, but by elevating the face so as to draw the trachea upwards, it is claimed we will obtain a murmur over the sternum when the bronchial glands are enlarged. I have resorted to this procedure in a number of cases and in only one was I able to get a murmur and unfortunately that case passed from under observation and I do not know whether enlarged lymph nodes were present or not.

DR. MILLER.—We cannot possibly get any symptoms unless the lymph nodes are large enough to compress the vessels or nerves or cause dulness. The symptom Dr. Dorning speaks of, upon which I believe great stress was laid by Eustace Smith, I have tried in a large number of cases, and it seems to me is of little value, as a murmur elicited by pushing the head back and putting the face on a level with the horizon, is present in many normal cases. If the vessels are put upon enough of a stretch a murmur will be produced. I believe with Dr. Rotch that there is absolutely no diagnostic symptom of value, unless the lymph nodes are very large.

DR. BLACKADER.—We all recognize the fact that in the diagnosis of enlarged bronchial lymph nodes, physical signs as a general rule give us very little assistance. On two or three occasions, however, I have noticed symptoms which led me to suspect an interference with the entrance of air into one of the bronchi, as evidence by a weakened inspiratory murmur, diminished expansion of the chest wall on the same side, and sometimes a recession in the lower intercostal spaces. These symptoms persisted for some weeks and were attributed by me to pressure exerted by enlarged bronchial lymph nodes as no other symptoms arose to more adequately explain them, and they gradually subsided under treatment. I am fully aware. however, how easily such symptoms may be misinterpreted.

DR. FRUITNIGHT.—I have seen a large number of *post-mortem* examinations in which tuberculous nodes were present, but nothing was determined clinically bv this manœuver of raising the head and extending the neck. So I think we have nothing positive in this method of examination.

DR. WEST.—I reported to the Society last year seven cases which I believed, and still believe, were enlarged bronchial lymph nodes. They were seen on account of cough principally, and the symptom Dr. Dorning has referred to was present in all the cases. Of course that symptom can be brought out in a good many children in whom there are no other symptoms of bronchial nodes. I have seen that in two other cases since my

paper was read and the symptom was prominent in them, and also in one of the reported cases with a recurrence of the trouble. The reappearance of this symptom in this one case was due no doubt to enlargement of the nodes. The enlargement of the veins of the upper part of the chest has been referred to, and that I found to a lesser extent in the face in some of my cases, and also some edema of the face. In four of the cases I have reported there was also a weaker respiratory murmur on one side than on the other. In a case that I saw last winter and again this winter, the child had a cough that resembled to some extent whooping-cough, and one other case seen this winter also resembled whooping-cough.

DR. DORNING.—I would like to ask whether either Dr. Blackader's or Dr. West's cases came to autopsy.

DR. WEST.—No; none of my cases.

ENTERIC FEVER IN CHILDHOOD.

BY A. D. BLACKADER, M.D.,

Montreal, Canada.

Typhoid fever as met with in children under fifteen years of age presents some characteristics which distinguish it from the disease as met with in the adult. These points of difference have already been referred to by several members of the American Pediatric Society.

During the past five years several outbreaks of this disease have occurred in Montreal, due in two instances at least, to the infection being conveyed in milk. Quite a number of children suffered, but a comparison between the numbers of children and of adults who were attacked I am unable to make, owing to defective registration of all the cases of the disease which occurred.

For this period I have the notes of twenty-nine cases of typhoid fever, the greater number of which occurred in my own private practice, but which include a few cases seen in consultation with other physicians. I have also examined the records of forty-eight cases treated in the Montreal General Hospital during this period, many of which occurred in my own wards; others I report by the courtesy of my *confrères;* also the records of twenty-three cases admitted into the wards of the Royal Victoria Hospital, the notes of which were kindly placed at my disposal by the attending physicians; making in all a total of one hundred consecutive cases occurring in children under fifteen years of age.

I have thought that a brief *résumé* of the characteristics of the disease as manifested in these cases, and of the relative frequency of the various symptoms and of the results obtained by

treatment, might present some points of interest to the Society. Of these one hundred children

1	was	under	the	age	of	1	year	7	were	under	the	age	of	9	years
3	were	under	the	age	of	2	years	10	"	"	"	"	"	10	"
3	"	"	"	"	"	3	"	9	"	"	"	"	"	11	"
5	"	"	"	"	"	4	"	9	"	"	"	"	"	12	"
5	"	"	"	"	"	5	"	8	"	"	"	"	"	13	"
8	"	"	"	"	"	6	"	8	"	"	"	"	"	14	"
6	"	"	"	"	"	7	"	9	"	"	"	"	"	15	"
9	"	"	"	"	"	8	"								

Four infants were thus under the age of two years, thirteen between two and five years, forty between five and ten years, and forty-three between ten and fifteen years.

Of the four cases under the age of two years, one was received into the Montreal General Hospital under the charge of Dr. Finley; two occurred in private practice; and one was seen in consultation.

In the case of the one received into the hospital the diagnosis was at first doubtful. The history was that of an infant of thirteen months, apparently healthy, with the exception of a perforated drum membrane, the result of an attack of scarlet fever seven months previously. A discharge from this ear persisted. Five days before its entrance into the hospital diarrhea set in, the infant became listless, fretful and drowsy, and food was refused. Two days afterwards it was seen by a medical man and recommended for admission into the wards. Its condition at the time resembled that of a child suffering from incipient meningitis. It was restless, turning from side to side, and moaning; its face had a cyanotic hue; the abdomen was slightly distended; no rose spots were visible; the spleen was distinctly palpable; the lower edge of the liver could be felt; a few moist râles were heard at the base of both lungs; the pulse was rapid and very weak; the heart was normal; loose fecal movements occurred four or five times in the twenty-four hours. The infant died in the morning of the fifth day after its admission. The Widal reaction was absent. The *post-mortem* examination revealed typhoidal lesions and the presence of typhoid bacilli in the intestines.

Two cases occurred during the spring of 1897 in my own practice. In both cases other children in the family were at the time suffering from well-marked symptoms of typhoid fever. Infection in these cases had been conveyed through the milk.

They did not run a severe course. The temperature ranged between 102° F. and 104° F. for the first week. In the second week between 100° F. and 102° F., but subsided to normal before the close of the third week. Rose spots were distinct in one, absent in the other. In both, the spleen was enlarged; in both, loose movements of the bowels were present, but the diarrhea was not sufficient to call for special medication. The only treatment employed was tepid baths at a temperature of 95° F. reduced to 90° F.

In the fourth case I was called to see an infant of eighteen months suffering from cerebral symptoms which the attending physician regarded as probably due to tubercular infection. At the consultation, a few rose-colored spots were discovered on the slightly distended abdomen, the spleen was found to be enlarged, and three or four loose movements of the bowels had occurred each day since the onset of the sickness. A probable diagnosis of typhoid fever was made, which I was afterwards informed had proved correct. After an illness of sixteen days an uninterrupted convalescence set in.

Typhoid fever in the infant is generally regarded as a comparatively rare affection. Marfan states (*Traité des Maladies de l'Enfance,* Grancher, Paris. 1897. Vol. i., p. 332), that it is remarkable for the vague character of the clinical picture and its difficulty of diagnosis. The more exact methods recently placed at our disposal for the determination of the presence of the typhoid bacillus will remove the difficulty in diagnosis; and all cases of continued, perhaps it would be better to say, remittent fever in the infant, unaccompanied by any distinct localization of disease, should be carefully investigated. My personal belief is that instances of this infection will be found more numerous than the facts elicited in previous discussions on the subject in our Society would lead us to think.

The statistics of typhoid fever in infancy are still too meagre to enable us to draw any broad conclusions. Thus far only the more severe cases have been recognized. With the more accurate means of diagnosis now at our disposal, the typhoid fever of infancy may be shown to run a comparatively mild course.

After a careful investigation of the records of those cases occurring after two years of age, I do not feel inclined to draw a dividing line at any special age. While in patients over fifteen years, the disease generally assumes the characteristics met with

in the adult, in my experience up to the age of fifteen it maintains the type met with in childhood; the symptoms are milder, and the duration in the great majority of cases is under three weeks.

In thirteen of my cases, the *onset* was sudden. Children apparently in good health were suddenly taken ill, so that within a few hours symptoms of disease were well-marked. In every case in which I have noted this fact, the sudden onset was associated with the disturbance of the gastro-intestinal tract, attributed at the time to an indiscretion in diet.

Of the well-recognized *initial symptoms*, headache was observed as present in 68 cases, (or 83 per cent. of the children over six years of age.) It is noted as severe in 16, (or nearly 20 per cent.) Vertigo is noted in 19 cases (22 per cent. of those over six.) Anorexia is noted in 49 cases. While no distinct chill is reported, in 12 cases the patients complained of a feeling of chilliness. In 18 cases vomiting is said to have taken place, but did not occur after the first day. Movements of the bowels, looser and more frequent than normal, were noted in 36 cases. Of these, 10 cases were distinctly diarrheal in character. Six of these were children in whom the sudden onset was attributed to indiscretions in diet. In only 4 cases did the diarrhea persist and require special medication. Constipation was present in a more or less pronounced degree in 59 cases, requiring rectal injections. Slight fulness of the abdominal parieties was noted at the onset in 48 cases. In 29, it is distinctly stated that no distension was present. Abdominal pain was noted as a complaint in 33 cases, while pain on pressure, a dubious symptom always in young children, is only stated to have been present in 15. Epistaxis occurred in 23 cases. Tonsillitis was present in 6 cases. A slight convulsion was stated by the mother to have occurred at the onset of the attack in an infant of two years and eight months, but as this was one of the instances in which, apparently, the sudden onset was precipitated by injudicious feeding, it has probably little value as an indication of typhoid fever infection. The personal equation enters so largely into any estimate of the value of these initial symptoms that it is impossible for us to draw conclusions from them as to the prognosis of the attack.

Investigating the symptoms occurring during the course of the disease, we observe that the temperature range presents

some peculiarities worthy of notice. A resemblance to Wunderlich's ascent at the onset was observed in only 8 out of the 100 cases. This small number is doubtless due to the fact that the temperature in hospital cases, and, indeed, in private practice, is rarely accurately recorded before the fourth or fifth day of the disease. Three of these 8 were cases in which the affection appears to have been contracted in the hospital, and as the temperature records were systematically registered, in them the step-like ascent is distinctly noticeable, in 1 for three, and in 2 for four days. After the first week, in the large majority of cases, the temperature became in a marked degree remittent. In those who were admitted into the wards towards the close of the first, or during the second, week of the attack, the temperature was remittent from the outset; a fall of from two to four degrees being recorded in the morning, as compared with the record of the previous evening. During the third week, these extreme ranges (in 62 out of the 87 charts at my disposal) came to an end, either gradually subsiding or more or less abruptly ceasing; so that at the end of the twenty-first day there was an evening temperature of not higher than 99 degrees. More frequently in the child than in the adult do we find the temperature at the close of this period remaining persistently subnormal for some days. In five cases it is noted that the rectal temperature remained between 96° F. and 98° F. for from three to four days. In one case for four days in succession, it recorded 95.5° F. as a morning temperature.

Of the 87 temperature charts which I have been able to compare, in 19 the temperature on several occasions reached or exceeded 105 degrees, and the fever persisted for four weeks or more. In 37 the temperature on several occasions reached 104 degrees, and the duration of the fever was about three weeks. In 15 cases the duration of the fever was between two and three weeks, but the highest range of temperature was 103° F. In 16 cases, while the temperature may have occasionally reached a high point, the duration of fever was under two weeks. Of the remaining 13 cases, the temperature charts are either wanting or too defective to make use of them, but of these, 4 I have characterized as severe in my notes taken at the time, and 9 as moderately severe. Taking the temperature curve, therefore, as some indication of the severity of the disease, I may refer to 23 of my 100 cases as being severe, 46 as moderately severe, and 31 as running a moderately mild course.

The pulse in the great number of cases was only moderately quickened, but in the few instances to which I will refer later on, it was rapid and dicrotic.

The spleen is noted as palpable in 70 cases. In 8 additional instances, the splenic dulness was noted as increased under careful percussion. Tenderness on pressure over the spleen is noted in 18 cases. Rose spots were noted in 55 cases. In three only are they said to have been numerous. A diffuse erythema of the neck and chest is noted to have occurred during the first week in 2 cases.

In 8 cases during the course of the illness the abdomen is stated to have become distinctly distended. In 5 of these, diarrhea was present. In two cases rigidity and tenderness existed, which subsided on the application of an ice bag. In 4 cases, 2 of them under ten years of age, traces of blood were observed in the stools between the eighteenth and the twenty-third days of the disease, but no severe hemorrhage occurred.

In 19 cases sonorous and sibilant râles are noted to have been present at the bases of both lungs. In 1 case a child of seven years is stated to have attended the out-patient department of the hospital for six days with symptoms indicative of an attack of bronchopneumonia. The physical signs noted were an impairment of resonance at both bases with numerous submucous râles; sibilant and sonorous râles over the upper portion of both lungs; and distant tubular breathing at the lower angle of the right scapula; temperature 103°; pulse 112; respiration 44. After admission into the hospital the spleen was found to be enlarged, and two days later, an eruption of rose spots occurred on the abdomen; the temperature assumed a remittent character, and the lungs cleared. Complete defervescence took place on the sixteenth day of the fever, followed by a relapse on the twenty-third day of the attack, lasting eight days. The temperature then fell to normal and convalescence ensued.

At the onset of almost all these cases, and throughout the attack in cases of moderate severity running a regular course, the pulse remains slow even under the stimulation of a high temperature, indicating possibly some action on the pneumogastric centre by the toxins of the typhoid bacillus; in severe cases, however, this action would appear to be more than counteracted by the effect of the toxin on the muscular wall of the heart, as indicated by the frequent development in children

of a soft, systolic murmur heard frequently both at base and apex. Its presence is noted during the second or third week in twenty-two of my cases. At the same time in three cases murmurs evidently more organic in character were also reported.

A mild nocturnal delirium is noted as present in eighteen cases. In only one instance was the delirium noisy. Restlessness in sleep, or sleeplessness, occurring during the second or third week is noted in 15 cases. In 12 cases drowsiness was a marked feature of the first week; and in 4 cases a condition of semi-stupor existed during the first few days after entrance into the hospital. In 1 case, with a dicrotic pulse there was muttering delirium, picking at the bed clothes, and subsultus, with a temperature of 105.5°. These symptoms fortunately passed off under free stimulation, continuous spongings, and the application of ice over the precordium. In a second case, in addition to the condition of stupor, a coarse tremor of the fingers was noted, and abolished reflexes. When convalescence set in a paretic condition of the muscles of the leg with dragging of the toes was noted. I have no record of the occurrence of temporary aphasia, instances of which have been reported by other writers, but Dr. Finley told me that in one instance this condition had been present for two weeks, but passed off completely during convalescence. A paretic condition of the bladder, requiring the use of the catheter, was noted in three cases during the second and third week of the attack. One instance of tenderness of the toes was recorded.

In only five instances is it stated that a trace of albumin was present in the urine; in two of these a few epithelial casts were also found.

Otitis occurred in four cases.

A benign non-suppurative periostitis is noted in one case.

A tendency to subsequent furunculosis was noted in two instances.

Relapses have been noted in 15 of the cases. In 1 case there were two distinct exacerbations. Relapses followed after both severe and mild attacks. It is stated by Marfan (*Loc. Cit.*) that they may be foretold by the persistence of the enlargement of the spleen, by the temperature failing to assume a normal and regular course, by the failure of the tongue to clean, and by the facial expression. I have failed to verify any of these statements, beyond noting that in 5 of the cases, the exacerbation

occurred in the fourth week after a severe attack before the temperature had quite assumed a normal range.

In corroboration of the value of the Widal reaction in diagnosis, I would state that in 43 of my cases it was carefully sought for with the following results:

3	gave	it	on	the	4th	day	1	gave	it	on	the	14th day
2	"	"	"	"	5th	"	1	"	"	"	"	15th "
2	"	"	"	"	6th	"	2	"	"	"	"	16th "
3	"	"	"	"	7th	"	2	"	"	"	"	17th "
2	"	"	"	"	8th	"	3	"	"	"	"	18th "
1	"	"	"	"	9th	"	1	"	"	"	"	19th "
3	"	"	"	"	10th	"	1	"	"	"	"	20th "
5	"	"	"	"	11th	"	1	"	"	"	"	21st "
4	"	"	"	"	12th	"	2	"	"	"	"	22nd "
3	"	"	"	"	13th	"	1	"	"	"	"	28th "

In 3 cases the reaction failed.

We may simplify these figures by stating that 12 gave the reaction on or before the eighth day; 13 gave the reaction after the eighth but before the twelfth; 12 after the twelfth but before the eighteenth; and 6 after the eighteenth and before the twenty-eighth.

The only death which occurred in this series of 100 cases was that of the infant thirteen months old which was received into the hospital in a condition of profound depression of the circulatory and nervous system, and died on the fifth day after admission.

The treatment of the majority of these cases was by the regular systematic employment of cool or cold baths. In 53 cases the bath was employed whenever the temperature rose above 102.4° F. In 19 instances the first few baths were given at 90° F. reduced to 85° F. and afterwards continued at 85° F. reduced to 75° F. Their duration was ten minutes; and they were repeated every three hours if the temperature remained high. In 25 instances the first baths were given at 85° reduced to 80° and afterwards continued at 80°, reduced to 75°. The duration of the bath was ten minutes; and it was repeated if necessary every three hours.

In nine instances after a few baths at slightly higher temperatures, the bath was given at 75° F. reduced to 68°.

In thirty instances systematic spongings with water at a temperature of 65° to 70° F. were given every two or three

hours associated with the employment of ice applied either to the head or in four instances to the precordium.

In three instances a cold pack was employed.

In fourteen instances the treatment was merely symptomatic.

Stimulants in the form of either whisky or brandy were employed cautiously in some instances, but freely in a few cases, regarding them not only as a stimulant, but as a rapidly and easily appropriated food. Strychnin was the chief cardiac stimulant employed. Milk formed the chief dietary, but a careful watch was kept over the stools that the quantity given should not be in excess of the digestive powers of the child. Frequently it was more or less modified by the addition of some diluent.

In regard to the use of cold baths and the Brand method in the treatment of typhoid fever in children, perhaps a short expression of my opinion may not be out of place. I am convinced of the great value of the regular and systematic employment of the cool or cold bath in the treatment of this affection. In my opinion it should be employed regularly without too rigid adherence to Brand's rule of only using it when the fever reaches 102.4° F., and a great fall in temperature as the result of its employment is not to be desired. Rapid falls, as we all know, are almost invariably followed by an equally rapid rise. As a recent writer has said: baths are to be employed for their action not on the temperature, but on the nervous system and through it on the heart, respiration, and secretions, especially the secretion from the kidneys. The nervous system of the child responds more quickly and energetically to the cool bath than does that of the adult, and the amount of response has to some extent an inverse proportion to the age. It is therefore unnecessary and undesirable that as low temperature should be employed in the case of a young child as in the case of an adult. The duration of the bath, the temperature of the water, and the frequency with which the baths are employed should be modified to suit each case in the same way as we modify the dosage of other therapeutic remedies. All sudden and severe shock should be avoided. I believe it to be a great shock to a young child to plunge it at the outset into a bath of 68° or even 75° F.; while a bath of 90° F. cooled to 85° and repeated regularly for the first few days of the attack gives rise to neither resistance, nor signs of shock or collapse on the part of the child. Later on in the disease lower temperatures may be employed if found

necessary. Even after the pyrexia falls below 102° F., I believe that the regular use of the cool bath once or twice a day strengthens the heart action and tends to a more rapid convalescence.

236 MOUNTAIN STREET.

DISCUSSION.

DR. COTTON.—I should like to ask Dr. Blackader if in these cases he administered laxatives or whether after having secured evacuation of the bowels by enemata, he noticed a subsidence of the temperature.

DR. BLACKADER.—The ordinary glycerin and water enema is almost daily employed in the hospital, rarely soapsuds, and any laxative influence is secured with small doses of calomel. I did not notice any special subsidence of the temperature.

DR. COTTON.—In 1885 a hundred tabulated cases were reported by me and reduced to percentages after the manner presented by Dr. Blackader. In these cases there was a very marked difference in the temperature ranges between the constipated cases and the cases with relaxed bowel. In that tabulation it was easy to see that after a movement of the bowels from enemata a decided fall in temperature occurred. From that time it has always been a matter of interest to me to note the effect of free movement of the bowels as compared with constipation. The paper deserves commendation for the carefulness in detail. I feel like corroborating, as the result of my own observation, the statement that in young children and older infants there is a tendency to subnormal temperature during convalescence and during the last week of the fever, when we have those oscillations, the temperature often goes below normal to rise again later.

DR. ADAMS.—That infants have typhoid fever I do not believe any of us will deny at the present time. I have seen infants under one year of age with typical typhoid fever; I have seen them at the ages stated. The point I wish particularly to discuss is first the fever itself, that is the elevation of temperature. In infants the rectal temperature is certainly the only correct temperature; and it is better to rely upon this method in all children. The temperature taken under the tongue in a child under twelve years of age does not register the true temperature of the child. So far as the nervous disturbances are concerned, each individual is to be studied separately. Different individuals, even in the same family, will present various manifestations. You cannot judge how a child is going to act with a given temperature. I have seen a rectal temperature not exceeding 102° F., accompanied by the most severe nervous

manifestations, and at the same time I have seen a temperature running six weeks at a high range with almost no nervous manifestations whatever. I have already reported to this Society cases of severe post-typhoid mental conditions, insanity, in children, and I have had two cases since the report was made of marked insanity following typhoid fever.

The method pursued by Dr. Blackader in the use of the bath seems to be the most rational one to employ, that is he judges his case, and employs hydrotherapy as indicated. We should not put every child in a bath. I put a child in a bath if necessary, not so much, as has been said, to reduce the temperature as to allay nervous irritability and improve the nervous condition. I think a great deal of harm may be done by placing an infant or child in a bath at too low a low temperature, some 20 or 30° lower than the body heat. Better results can be obtained with a temperature 15 or 20° less than the fever, and not so great a shock given as to require free alcoholic stimulation. Next comes the food. If a child will not take the food ordered for it, that is, usually milk or broth, ice cream may be given. Ice cream is not the ideal diet for children, but in a case under my care in which something had to be done, the ice cream was tried and the child recovered. The child had a rectal temperature of 105 to 106° F. for two weeks. I could not induce the mother to apply hydrotherapy. She would not have a trained nurse, she and the father insisted upon taking care of the child and it was under the most unfavorable circumstances, but it did seem to do well with plain vanilla ice cream.

DR. NORTHRUP.—I wish to state that I think it has been proven that children under two years of age are little susceptible to typhoid fever. Under repeated exposure, as in epidemics, they may acquire it. I wish to repeat what I have said before, and I think the work done in Boston proves it, that infants under two years of age are not susceptible to the poison of typhoid. Now as an exception, I have recently had a case under two years old, but it was the sixth case in the same family. First an elder child, then the father, and finally an elder brother had the disease. The mother was the only one well. This child for three weeks was sucking its thumb and wiping it over the face of the father. That baby was nine months old. Another case fourteen months old was a nursed baby. The father thought it was very cute to have the baby eat with him his noonday meal out of his bowl of milk. I have seen seven cases under two years, and every one of them has had the history of repeated prolonged exposure and their immunity seemed to be overcome by the overwhelming exposure of an epidemic. It is not an infant's disease. Now, another point, when they do have it, is there anything mysterious about it? Do they have a prolonged fever that is difficult to detect? I maintain that they do not.

The infant of nine months was one of six exposed to infection for three weeks. One was so far along it did not count, but it was one of four in the hospital, and all reacted to the Widal test. This one was late in reaction. She had the Widal test only on the first day of convalescence. But tympanites, rose spots, Widal test, enlarged spleen and diarrhea were present. There was nothing mysterious about it at all; it had all the characteristics of the disease. One of thirteen months had also all the characteristics. It had the profound and sunken appearance, the pallor and rose spots, and all the common symptoms of typhoid fever in infants. Enlarged Peyer's patches do not always mean typhoid fever in infants. The men who did work twenty years ago would not accept that as pathognomonic; and, neither will I accept any little, mysterious fever lasting two or three weeks as typhoid fever in children. Most of the doubtful cases are simply cases of enterocolitis. It is not so doubtful as in adults, for in adults a three or four weeks' fever is very apt to be typhoid fever, and that does not apply to infants; and neither will I accept the enlarged Peyer's patches as evidence that infants have had typhoid fever.

In regard to treatment, any temperature that is too high in children I cool with water. I give no antipyretics. The only question is how to cool them. I usually advise cool water inside and cool water outside. I am afraid of the tub bath in children, because they usually struggle against it. Use cold water, lay cold cloths about these patients, and give cold water internally. That was the only treatment we used in the case to which I have referred, except twice, I believe, we used the tub. I always use heat to the feet, cool water to the trunk, and apply cold water to the head. The child nine months old we fed on modified milk from the Walker-Gordon laboratory. We have a record of what it would take and what it would not take. It would take 3 per cent. fat, 6 per cent. sugar and 1 per cent. proteids, with 10 per cent. of lime-water, and would take nothing else. We could not reduce the lime-water or increase the proteids, but it would take the proportions I have given freely. I like ice cream, but often we cannot be sure it is clean cream. It is better to give a good corn starch than dirty cream.

DR. GRIFFITH.—I must say that I take issue with the last speaker entirely. It seems to me that his position is not altogether logical. He will admit that if an adult should have a continued fever for which we cannot find any cause, the mere fact that it is continued makes it probable that it is typhoid fever, even although there are no definite symptoms of the disease discoverable. On the other hand, if the case is in a child, he insists that he must have tympanites, and rose spots, and diarrhea, and all that, before the diagnosis of typhoid can be made; and this, too, in spite of the fact that children, even

more commonly than adults, fail to show a typical picture of the disease in spite of its certain presence. My experience is that typhoid fever is of a frequent occurrence under the age of two years, and that it is frequent even under one year, but that it is difficult to recognize. It is thus difficult to recognize, because infants so frequently have continued fever from so many different causes, and we are, therefore, prone not to have the idea occur to us that the disease may be typhoid; while in an adult we at once consider that this may be the nature of the trouble. Formerly the cases were called remittent. Now they are supposed to be enteritis or gastritis or auto-intoxication, and so on, when really they are typhoid in many instances. I am convinced that if we were to examine these infants more carefully we would more often find rose spots and enlarged spleen. It has been my unfortunate experience, as a resident of Philadelphia, to see a great deal of typhoid fever in infants and children. For a while last year the wards of the Children's Hospital were so full of these cases that it was difficult to get cases enough of other diseases to present to the class.

I have repeatedly seen the disease in children under two years of age. Often in consultation I have seen it overlooked by the physician in attendance, who ought to have recognized it, for the spots were there in full force, and he would certainly have seen them if he had looked for them. I can recall cases seen at three months, at seven months, at nineteen months, and so on. Of course, nobody disputes the fact that typhoid fever is *comparatively* uncommon in infancy, *i.e.*, as compared with the frequency of the disease in adult life; but that it is *actually* rare, or that children are not likely to get it if exposed to the infection, I do not believe. Certainly, the experience of many physicians shows that this rarity is not the case. This has become especially evident since the serum reaction has come to our aid.

I would like to dwell particularly upon what has been said about the use of water. There are many cases, as far as my experience goes, in which bathing does not answer well in children with typhoid fever. I have repeatedly given up first tub bathing and then sponging, because I found the child's condition was made worse rather than better. I think it may be safely stated that children with this disease do not bear hydrotherapy as well as adults do, as a rule. In the Children's Hospital we do not give the baths below an initial temperature of 85°, and often they are given at that temperature without any effort being made to cool them later. Often we find that even 90° or higher does not answer well.

DR. ROTCH.—What Dr. Griffith says about children is very true. Dr. Northrup, as I understand it, refers to cases under two years of age. I think all the evidence points towards the possibility of young infants being affected by typhoid. The

reported cases are increasing all the time. We have had 16 cases which have been proved to be typhoid, and 8 of these died. Although the spleen is probably always enlarged in these cases, it is not possible always to detect it. It may be that infants are not as susceptible as older children and adults, but I think it is a pity to say so much about it, for I think they are susceptible; but infants are not so likely to receive food by the mouth carrying the typhoid bacillus as the older children are. The cases of Dr. Northrup were practically fed upon typhoid bacilli, and it is no wonder they had typhoid fever. Those that get the typhoid bacilli have the disease, and those that do not get them do not have it. I am familiar with Dr. Northrup's work; but I do not think that the pronounced symptoms of typhoid fever appear in early life as they do later. The cases with the severe symptoms are not the common cases in early life at all. Out of hundreds of cases in the Children's Hospital, we have had some severe cases, but usually the cases are exceedingly mild in infancy and early childhood. It corresponds more to the aborted type in adults, and the lesions usually are not nearly so pronounced at autopsy. The rose spots, I think, will be found more common than is usually suspected. We have had a large number of cases in the hospital in the past year. In our experience warm baths or sponging is effective. A very good way is to wrap the child in warm moist gauze and then fan it, which reduces the temperature, and is usually very satisfactory. Children with typhoid fever almost invariably get well, except in severe cases, such as Dr. Northrup referred to, and those cases usually will die. The cases I have referred to gave the Widal test and presented undoubted symptoms of typhoid fever.

DR. WILSON.—I believe no inference regarding treatment can be drawn from a series of 100 cases. But Dr. Blackader's cases were treated by several methods and not according to any one special plan. I note several points in the paper that I would allude to. First, the fact that in this series of 100 cases there were but 4 cases under two years and 1 case under one year. That gives 1 per cent. under one year and 4 per cent. under two years. I note also the fact that the occurrence of relapse was 15 per cent., which is interesting, since the statistics upon relapse are very unsatisfactory and vary very much. The two reasons that led me to speak are first, the importance of emphasizing some facts brought out by this discussion. First, as to the predisposition of age. I entirely concur in what Dr. Northrup has said, namely, that infancy up to two years carries with it a certain immunity as compared with the next few years, and a high degree of immunity as compared with adult life. I have been in the habit of pointing this out to the students and calling their attention to the fact referred to by Dr. Rotch, that this immunity in all probability is not an essential immunity but an

accidental relative immunity. That is to say, the infant in arms is not exposed to the liability of infection to the same extent that the older child and the adult are exposed. I think that is the only philosophical way of looking at the matter from the data that have been collected.

In reference to the diagnosis of enteric fever in infancy, it seems to me we have to recognize two types of enteric fever if we want to have our knowledge conform to the facts. One of them is the infantile type and the other is the adult type. The adult type occasionally occurs in infancy, and the infantile type not infrequently occurs in adults. Perhaps I should not take up your time with this because it seems such familiar knowledge. The infantile type lacks the ordinary clinical diagnostic criteria of the adult type. The infant with typic enteric fever has spots which must be carefully looked for in a large proportion of cases; almost always a spleen which may be at some stage of the process palpated or may be made out by percussion; often diarrhea, rarely tympanites, and almost always a temperature range of remittent type. Curiously enough, the patient is often so little ill that you have difficulty persuading the parents that it has typhoid fever at all. In cases of the infantile type the symptoms are so mild that you often cannot persuade the mother that the child has enteric fever, although the evening temperature may reach 103° or 104° F. Again typhoid fever of the infantile type is not only a disease in which the symptoms are mild, but also in which the prognosis is correspondingly favorable. Dr. Blackader's cases show a mortality of only 1 per cent., although the series include many cases of the adult type. Thus we have a mortality altogether lower than in the adult in enteric fever.

It seems entirely proper that I should say a word upon the subject of hydrotherapy, particularly in the treatment of enteric fever in childhood. In the German Hospital at Philadelphia we have a children's ward in which are treated many cases of enteric fever. In fact, I collected and analyzed two or three years ago 150 cases which I have never had the opportunity to prepare for publication. We treat the cases there by systematic cold bathing, according to the method of Brand, with certain modifications. That is to say, systematic bathing according to that method is followed in a general way, but no humane doctor will treat every case upon the same plan; although the plan is a routine procedure, we often find it necessary to vary it. We commence with a bath of 85°, 90°, or 95° F., according to the intensity of the febrile movement and the state of the secretions and the general condition of the child, and then by gradually lowering the temperature of the successive baths we find what degree of cold the child will bear, and this determined, we carry out in general the formula of Brand as to the temperature. Thus by the adaptation of the plan to the individual we obtain results

which seem to us most satisfactory. The children actually come to like their baths. The bath is not cooled by the addition of ice or cold water after the child is in it. The duration of the bath is from eight to fifteen minutes. The next bath is given a little cooler, until we get the temperature the child bears with comfort.

The Brand method is not essentially an antipyretic treatment, and one of the difficulties in the way of its general acceptance arises from a failure to understand this cardinal fact, which Brand and his followers have always insisted upon. The antipyresis is only an incident of the treatment. The rhythmical repetition of profound impressions upon the nervous system by means of the application of the bath constitutes a means of modifying physiological processes that are disturbed and pathological processes that are in progress, which reacts favorably upon the nervous system, incidentally upon the temperature, upon the secretions, and finally upon the nutrition, thus constituting, when all the factors are taken together, a positive useful treatment of typhoid fever, capable of decidedly and constantly reducing the mortality.

Why are not little children given the advantage of the cold bath or cool bath? There are two objections. First, the nervous system of children is more susceptible; and, second, the superficiaries of the child are greater in proportion to the bulk of the body than in adults, and there is a danger of over-chilling the child. Therefore, every thoughtful clinician who has practiced it at all knows that the method of Brand cannot be practiced in the child in the same manner as in the adult.

The temperature used by Brand is 68°, and usually we use in adults 68° to 70° F. In children we begin with a temperature possibly of 90°, and gradually use colder baths until we reach 80° or 70°. The child is gradually lowered upon a blanket into the bath, and it does not get the violent shock which we like to get in the adult—a shock causing deep breathing.

DR. FRUITNIGHT.—I wish to speak of a case in which there was an intercurrent attack of measles. A boy, aged seven, had severe headache, temperature 102° to 103° F., and irregular meteorism, tenderness on pressure, enlarged spleen, rose colored spots, and the case gave a positive Widal test. His sister came down with measles and afterward some other children in the house also had measles. On the thirteenth or fourteenth day of his typhoid, the boy had a temperature of 105°, with the symptoms of measles, including the exanthem, as a consequence his delirium and prostration were increased, and after the measles had run its course, as it usually does, the enteric fever continued its regular course.

I have never before seen this coexistence of these two affections and I think it must be very uncommon. I would have doubted the occurrence and presence of the measles in this case,

had not the other children of the house been attacked with measles, to whom the infection of the typhoid fever case could be directly traced.

DR. GRAHAM.—As I understood Dr. Blackader, the only death occurred at thirteen months, and I would like to know whether an autopsy was made in that case and especially what was found in the bowel.

DR. FREEMAN.—As to the susceptibility of children to typhoid fever, I believe important evidence may be obtained by a study of epidemics of typhoid fever due to the drinking of contaminated milk. In such epidemics it is found that as a rule the liability to attack and the severity of the attack are proportional to the amount of uncooked milk taken, and that those on an exclusive milk diet are most apt to suffer from the disease and have it most severely.

Should this rule apply to infants it is evident that during milk epidemics most of the cases and the most severe cases would occur in children under two years of age. As a matter of fact, in such epidemics children under two years of age are rarely attacked, and if attacked are not usually very severely ill.

DR. MILLER.—I only wish to suggest that one reason the disease is supposed to be rare under two years is because typhoid cases at this age rarely come to autopsy. It is much like lobar pneumonia which was formerly supposed to be very infrequent in infancy, but which clinical observation has shown to be quite common. That belief was founded upon the fact that it was not found often at autopsy; but it was because children rarely died with the disease but usually got well. And the same, I believe, is true in typhoid fever.

DR. DORNING.—Apropos of Dr. Northrup's remarks, I will say that one summer I had three children die of asthenia consequent upon chronic diarrhea. At the autopsies there was found in all 3 cases involvement of Peyer's patches, and the gross appearance gave the impression that they were cases of typhoid fever. The pathologist, however, reported them as cases of follicular enteritis.

Regarding tubbing, or the Brand treatment, my conversation with physicians who have themselves suffered with typhoid fever and who have been subjected to it has shown that, with one exception, they all protested against it. In the use of water I think there is room for great discrimination. After a study of hydrotherapy the more I see of it the more I am impressed with its value and also with its potency for harm. Out of a dozen persons sent to the seaside for bathing, 6 will be benefited and the others will be either injured, or not benefited at all. Cold bath is merely a relative term. Babies do not stand cold water well. A baby with a temperature of 105°

cannot be put into a bath of 68° without shock which will be appreciated in the bluish extremities and the disturbed heart's action. I believe it is best to begin with a higher temperature and gradually reduce the temperature of the baths One point that has been omitted in the discussion is that while applying water, either in the tub or by cold sponging, there must be kept up continual friction of the surface. This is of real value in the hydrotherapy of fevers. By it we counteract the depressing effect of the bath. My own experience leads me to give preference to cool sponging in children. I usually begin sponging with a temperature of 90° and gradually reduce the temperature.

In regard to diet, there is under discussion at the present time the administration of more substantial food to typhoid fever patients. This past winter I have had three typhoid fever cases who have received solid diet. For instance, one girl sixteen years of age, was brought in, who had received the routine diet of the family. She was in the second week, temperature 103° and the usual symptoms of typhoid fever. There was a slight diarrhea. She seemed to have progressed so well on the diet that I ordered little or no change. She did well; had no intestinal hemorrhage, no aggravation of the diarrhea, or any unfavorable symptoms. She remained in bed eight days, the temperature went down rapidly and five days later, under protest, went to her home. She seemed perfectly well and strong. Two other cases that were given substantial food during the third week made a good recovery without complications. In one case in which I attempted to give some solid food there was a subsequent rise of temperature. I did not know at the time whether that was due to the food or some complication. Later on there developed a large abscess over the trochanter, which probably explained the rise of temperature.

DR. NORTHRUP.—This discussion seems to have resolved itself into a question of personal experience. Now at the Presbyterian Hospital I have had as many as thirty cases of typhoid fever at one time. I have had that service year after year, so that I have at least looked at typhoid fever in my day. At the Foundling Asylum I have seen approximately two thousand autopsies on children, under three years, and in none have I, in my judgment, seen typhoid fever. You may say those are institution children fed on sterilized milk and no typhoid is allowed to get within the door. But eleven hundred of these cases are farmed out and feed on corner grocery milk. Eleven hundred of those are out virtually looking for typhoid fever, but they never bring it back. Dr. O'Dwyer and Dr. Smith and I have been watching for typhoid fever, but we have not found it. I wish to encourage a healthy scepticism in all indifferent fevers under two years. I do not believe children under two years are susceptible to typhoid fever, except feebly

so. Now when they do have typhoid fever they declare it. They have some definite symptoms. They must give the Widal test or have spots, in my estimation, to justify us in making a positive diagnosis of typhoid fever. The most common mistakes arise in early diagnosis, in misnaming the following diseases, influenza, subacute catarrhal enteritis, central pneumonia and malaria.

Again I want to endorse what Dr. Wilson said, and I presume everybody believes it, although I did not think to say it when I was speaking. We do not give the baths for the temperature alone; it is not for the fever, but for the nervous symptoms. And what I call a cold bath is not much below a hundred degrees when it is given for the nervous symptoms. It is for the nervous symptoms, for a heart tonic and for the secretions that we give the bath.

DR. BLACKADER.—I regret that some of the statements in my paper have apparently been misunderstood by one or two of the speakers. My remarks on the persistent low temperature sometimes noted in children only refer to the end of the third, or the beginning of the fourth, week after the period of intermittent pyrexia has passed off. Attention has been called to the frequency with which several children in one family are attacked with typhoid fever. My charts show the same fact, and in two instances in which the first case of the disease was not recognized, the infection was conveyed to many other members of the household, both young and old. This spreading of the infection is more liable to take place in the case of infants and young children than in the case of adults. Dr. Northrup refers to 2,000 *post-mortem* examinations of infants, and states that judging by the lesions found in the intestines, typhoid fever in infancy must be extremely rare. But Dr. Northrup himself admits that the lesions of infantile typhoid fever are ill-defined as compared with those of adult age. With our present knowledge, infection by the typhoid bacillus can only be excluded after a careful bacteriological examination with the newer tests. For this reason I cannot accept his inferences as conclusive. In reference to the employment in children of baths as a therapeutic measure, I desire to again emphasize, as Dr. Wilson has also done in his remarks, the necessity of modifying the temperature and duration of the bath to suit each individual case. The object to be obtained is a tonic effect on the heart and vasomotor centre. I do not think that warm baths, or warm baths followed by fanning, can exert the tonic or stimulating influence on the nerve centres, so desirable in a continued fever, and which are obtained from the regular systematic employment of cool or cold baths. At the same time I quite agree with those who deprecate the use of baths at such low temperatures as to frighten, or unnecessarily shock the child; a condition which

may in some instances antagonize all the good effects to be obtained from the bath. I may say that in my own practice, while employing cool baths in almost every case except the very mild ones, I have obtained only good results, and have heard no serious objection to their use, either from the little patients or their parents.

EXCLUSIVE SOUP DIET AND RECTAL IRRIGATIONS IN TYPHOID FEVER.

BY A. SEIBERT, M.D.,

New York.

To diminish the number of pathogenic organisms in the human body is to-day recognized to be the best treatment of infected persons. In typhoid this object can be attained successfully by following two distinct indications, namely: (1) by promptly removing all remnants of food from the alimentary canal, and then permitting only such articles of diet to come in contact with the infected surfaces of the intestine as will offer but poor culture media for the typhoid bacillus and its neighbors; and (2) by systematically irrigating the rectum during the entire course of the disease.

To give a cathartic at the beginning of an attack of typhoid is universally practiced, but also the early replacement of the infected alimentary contents by food which will even feed the remaining bacteria better than the removed solids, namely: by milk. This is like emptying a dish of decomposing solid food and immediately filling it again with fresh milk, in the expectation of keeping the latter sweet.

Until the chief cause of gastroenteritis in children had been demonstrated to be the manure bacteria that drop into the milk used for infant feeding, their rapid multiplication during warm weather and their action upon the children who swallow them, and until the imperative therapeutic necessity had been established to withhold all milk from an enteritic patient until he was cured, our treatment of gastroenteritis was but in a doleful state.

To my mind, patients fed on milk during an attack of typhoid (a specific form of enteritis) are but little better-off to-day than our former little patients were during an attack of summer-complaint, with milk and opium-mixtures in their intestines. If most of our typhoid patients fed on milk were not adults but children, like in gastroenteritis, the percentage of typhoid mortality of to-day would equal that of gastroenteritis of former

years. For not alone is the typhoid bacillus to be considered in the bowel of the typhoid patient, but also the many other alimentary bacteria that take part in the attack on the human organism during this disease. If milk is the best food of intestinal bacteria during enteritis without typhoid mixture, then I see no reason why it should be less favorable for their sustenance and propagation during enteritis caused by them *and* the typhoid bacilli.

Reflections of this nature caused me to try the possibility of feeding typhoid patients on a fluid diet not including milk. My first case so fed was under observation during October of 1889, and since then none of my typhoid have been given milk until the rectal temperature has been normal for at least two days. It was found that at the beginning of the attack that plain, cold water sufficed during the first twenty-four to forty-eight hours after the initial purge, although, of course, much depended upon the gastric condition of the patient. Then soups made of meat-broths, containing oatmeal, barley, rice, and peas, strained, of course, and well spiced with salt and pepper; and after another two days lentil-soup and the yolk of a fresh egg added to the oatmeal, rice and barley soups, were given, so as to allow an adult one-half of a pint of two kinds of soup alternately every three hours, and smaller quantities to children according to age. Five meals in all were given during the day. At night only fresh, cold water was given, *ad libitum*, as well as during day-time, in the intervals between the meals. Five to fifteen drops of the diluted hydrochloric acid were given before each meal, unless hyperacidity prevailed. No other medication was employed, irrespective of the height of the temperature or the frequency of the stools. Alcohol was but given in small quantities to habitual topers during the first few days, at night. Occasionally cold, strong, black, sugared tea was used as a stimulant.

In July, 1889, Backhaut, an assistant in Prof. Mosler's clinic at Greifswald, published a report (*Deutsch. Med. Wochenschrift*, July 18, 1889) on the treatment of typhoid patients by rectal infusions of one-half per cent. tannic acid solutions. The good results obtained were attributed to the germicidal action of the tannin. I ventured to suggest to Prof. Mosler, by letter at the time, that the twice daily executed cleanings of the lower end of the colon might be the cause of the observed beneficial effect

upon his patients; and from that time on every typhoid patient under my care was given two to four rectal enemata of plain warm water daily. It was soon found that rectal tubes were harmful and unnecessary, and that if the buttocks of the patient were but elevated upon the bed-pan, the water flowing gently from the fountain syringe, hanging about three feet above the patient, would dilate the lower colon sufficiently to dilute and carry off the accumulated typhoid feces. Furthermore a short tip introduced through the sphincter ani cannot possibly reach typhoid ulcerations in the descending colon. Since the summer of 1889 these therapeutic measures have been employed by me in every typhoid patient in private practice as well as during my ten years of service in St. Francis Hospital in New York. In all 153 cases were treated in this manner. Seven cases ended fatally, of which 3 were brought in moribund and four had complicating bilateral pneumonia.

RESULTS:

1. Delirium, headache, insomnia, nausea, vomiting and tympanitis usually disappeared within forty-eight hours of treatment.

2. Tympanitis, nausea and vomiting never developed in any patient, even when complicating pneumonia was present.

3. The fur on the tongue disappeared within a few days.

4. Appetite came frequently on the fourth day of treatment, even when the thermometer registered 102° to 103° F.

5. Even excessive diarrhea (fifteen to twenty-five daily stools) disappeared invariably within the first week of treatment.

6. In all uncomplicated cases the temperature began to decline within twenty-four to forty-eight hours after the beginning of treatment and invariably would reach the normal figure within ten to twelve days.

7. In cases complicated by pneumonia, nephritis or phlebitis when treatment began the temperature usually remained in accord with the inflammatory conditions found until these also disappeared, while the cerebral, gastric and intestinal disturbances usually subsided as rapidly as in the uncomplicated cases, excepting anorexia.

8. Complications, when not present at the start, were very rare and then usually developed within the first two days.

9. Intestinal hemorrhage was noticed in three cases, none ending fatally. Perforation did not occur.

FINAL REMARKS:

It was immaterial whether this treatment was begun in uncomplicated cases during the first, second, third or fourth week of an attack, for the above mentioned improvement always began within forty-eight hours, exactly like in gastroenteritis. Cases coming under treatment during the first two weeks of illness usually presented more marked and rapid improvement during the first four days of treatment than older cases.

That milk given to a typhoid patient will cause a new rise of temperature after days of improvement on a soup diet, I have demonstrated time and again to my house-staff in the hospital.

Many of the pneumonia attacks complicating typhoid are due to secondary infection through the blood by organisms finding their way to the lung tissue from the intestine, like in the systemic infection of enteritis in children. By diminishing the quantity of absorbable toxic material in the intestine by appropriate diet and frequent rectal irrigations, we cut short the supply for systemic and pulmonic invasion, and materially aid the restitution of normal conditions.

Typhoid bacilli will readily grow in soup, but this food is so rapidly absorbed that in comparison to milk curds it cannot aid their sustenance long enough to injure the patient.

During the last ten years I have alluded to this plan of treatment in three publications, in *Medical Record* of September 12, 1891; *New York Polyclinic*, March, 1893, and *Medic. Monatsschrift* of July, 1894. In the June number of the *American Journal of the Medical Sciences* of 1894, Prof. Yeo, of King's College in London, England, also called attention to the dangers of indiscriminate milk-feeding in typhoid. Other literature has not come to my notice.

114 EAST FIFTY-SEVENTH STREET

TWO CASES OF FATAL LEAD POISONING.

BY ALLEN BAINES, M.D.,

Lecturer on Diseases of Children and Associate Professor of Medicine, Trinity Medical College, Toronto; Physician to Victoria Hospital for Sick Children and Assistant Physician, Toronto General Hospital.

It is not with an idea of producing anything original that this paper is presented, but from the fact that clinical histories of lead poisoning in children of a sufficiently extended and minutely noted character are but few. Dr. Putnam in his comprehensive article on "Lead Poisoning" in *Keating's Cyclopedia*, says:

"The justification for the present article is found not in the abundance, but in the meagreness of the present accumulation of facts relating to children, and the importance of taking steps toward increasing it." There is no doubt that cases of lead poisoning in children are rare, the diagnosis not at all simple, and the treatment unsatisfactory.

My justification is somewhat on the same line, and I can bring but one symptom, unknown to me before now, into prominence, viz., a very pronounced dark blue circle about the anus, more marked from the fact that the children were of fair complexion, otherwise, there is nothing but a carefully taken clinical history and the fact that until the day of the first child's death, no previous history having been obtained, the case resembling so clearly that of basilar meningitis, it was treated as such.

Charles H., aged two years, eight months, admitted to the Hospital for Sick Children, November 20, 1899.

FAMILY HISTORY.—Father, living, healthy, not affected by the lead. Mother, living, healthy, until three weeks since, when she was taken ill with a sharp attack of lead colic, accompanied by the other classical symptoms found in these cases.

The other members of the family consisted of a brother aged five years, nine months, since dead from same cause, an infant at the breast, perfectly well, apparently untouched by the lead. The manner of absorption being, that for a week previous to the first child being affected, the meals had been cooked with

fire wood procured from staves of old barrels which had contained white lead, so the fumes of lead not only entered into the food, but permeated the atmosphere.

PREVIOUS HISTORY.—The child had never been ill with any complaint whatever, until November 6th, when he had a well marked convulsion, lasting about five minutes. He recovered completely from this attack and was quite well until the evening of the 19th, when he had four convulsions in a few hours' time, the duration lasting from three to six minutes. He has been practically unconscious since the first convulsion, rousing slightly at intervals for a few moments, and has had frequent attacks of vomiting; bowels for the past few days have been somewhat constipated, necessitating mild purgation; appetite ravenous, no history of worms having been passed.

EXAMINATION.—General inspection. He lies on his back in a semi-comatose condition from which he cannot be roused; eyelids half closed, eyes roll slowly from side to side, mouth slightly open, tongue moist and coated, breath nasty and offensive, the same odor seemingly to emanate from the whole body. The right arm and hand firmly flexed, the left extended at the side, occasional twitching of the right fingers of a spastic character. Respiration slow and irregular; abdomen markedly contracted, legs extended, but nothing abnormal. Lungs, normal; heart, first sound aortic accentuated—second sound pulmonary accentuated. Rhythm, irregular, intermitting. Pulse, irregular, full-soft. Eyes, sclera pale, but clear, pupils dilated but react and are equal.

Reflexes.—Plantar absent, patellar absent, cremasteric absent, abdominal absent, Kernig's sign absent.

Can rouse patient only by causing pain on pressure, when he utters a moan and sinks back into comatose condition. Temperature 99.3° F., pulse 60, respiration 14. The condition remained practically the same during the night; temperature rose to 101°, pulse 65, respirations to 20. A condition of meningitis being suspected, collodion vesicans was applied to nape of neck and ice cap to head—an enema was given, without any effect. Three convulsions occurred during the night, the first two involving the right side of the body, the third, the left—in all three the face and neck was violently convulsed.

November 21st.—Twenty-four hours after admission—9 A.M. Abdomen still more contracted, breathing slow, deep, entirely

thoracic, very irregular, intervals of six to ten seconds, occurring between respirations, then regular again. Tache cerebrale well marked. The eyes, examination showed marked neuritis, no choked discs, veins engorged and tortuous. During the day, eighteen distinct convulsions were recorded, most of them being general and epileptiform, but some were unilateral—sometimes only the fingers being involved. Patient still comatose, temperature 102° F., pulse 72, respiration 30, being more regular. Pupils now contracted and equal.

The following description of the features of the convulsions most marked:

First the face would get flushed, eyes would open wide, fixed and staring, pupils widely dilated. The eyes would then roll about the eyelids and lips begin to twitch, and soon whole sets of muscles, face, arms, legs and body; every convulsion beginning from above downwards, care having to be taken to prevent biting of the tongue and lips. During these attacks he gave vent to a peculiar, grunting moan; no opisthotonus.

November 22d.—For the next twenty-four hours there were no convulsions, not even twitching; he appeared much better, retained nutrient enemata—all food by the mouth being at once expelled. Respirations more rapid—34 free, deep and slightly abdominal, at times, however, becoming irregular or rather Cheyne-Stokes in character; pulse rapid—124, regular and soft.

November 23d.—Seemed better and possibly partially conscious. He kept licking his lips, and when asked if thirsty, would nod his head, swallow water eagerly and retain it, but about 3 P.M. began to vomit and kept it up incessantly, not with any effort, the vomitus simply running from the mouth. At 4 P.M. he became quite conscious, sat up and asked for his mother, but almost immediately sank back, lapsing into a comatose condition. The vomiting ceased and twitching began again, pulse went up to 130, respiration 34, temperature 103.3° F. He could not now be roused. For the first time eye symptoms were noticed, external strabismus of left eye with pupil contracted, right dilated and not reacting to light.

During ophthalmoscopic examination, he had a strong convulsion, head being retracted and drawn to the right side. After the cessation of the convulsion the left external strabismus had disappeared, and instead, there was right external strabismus

with the pupil still dilated. Ten more convulsions followed, they were slight, involving the facial muscles and right arm, respirations deep and irregular, pulse rapid, low tension. During the next two hours, he had twelve convulsions, nearly all general. The last one was simply a series following each other in such rapid succession as to be uncountable, and lasting for thirty-five minutes. One hour after the last convulsion the patient died, the cardiac centres failing first, the breathing going on in long inspirations, ten to twenty seconds apart for three or four minutes, after the pulse had ceased to beat.

A *post-mortem* examination was refused.

CASE II.—S. H., aged five years, nine months, brother of Case I., admitted to the Hospital for Sick Children, December 4, 1899. Previous history very good, no illness of any kind. Present illness for past three or four days had severe pain in abdomen, bowels obstinately constipated; this morning had a convulsion of entire body. Whilst waiting for admission, had a series of seven or eight convulsions in twenty minutes. These differed, some being general, some unilateral, mostly right side, and, as in the case of his brother, remained in a semi-comatose condition.

The history so nearly resembles that of Case I. as to make it unnecessary to repeat it. The child lived for nearly the same time to an hour. Knowing of the history of Case I., I had Prof. Ellis examine the urine, four ounces being sent him. He reported the quantity to contain four milligrams of lead.

The convulsions were many, ninety-five on the first day, eighteen during the night. Towards morning the convulsions ceased, but he gradually became weaker and died at 3 P.M.

The *post-mortem* examination revealed nothing markedly abnormal.

BACTERIOLOGICAL REPORT.

Post-mortem examination, December 7, 1899 (twelve hours after death).

Cultures, from kidney, spleen, liver, base of brain and lung, all on blood serum.

Coverslips, preparations from brain, show a large, thick bacillus, freely segmenting, ends rounded; also a diplococcus, the body of which is elongated, resembling the micrococcus lanceolatus; some pairs are enclosed in capsules, others not so.

December 8, 1899.—Cultures from kidney, liver and spleen show no growth. Cultures from brain show numerous small, raised, discrete colonies, white or pearly in color, with glistening surface.

Coverslips show short, elongated bodies in pairs, end to end, as in the original coverslip preparations, no capsules are seen about them. They are isolated in pure culture. The large rod is not found in culture. The reactions on different media are as follows:

Seventy-two hours old.

Bouillon, dense, clouding, stringy sediment.

Gelatin, growth of white colonies along the stab; no liquifaction.

Agar agar, diffuse, grayish film along line of smear; the edge is wavy and more dense than the central part.

Potato, slight but distinct, moist growth, white in color along line of smear.

Litmus milk, marked acid production and coagulation of casein.

Culture from the lung show (1st) a yellow raised colony; (2d) a white raised colony.

Colony 1, "the yellow colony," shows staphylococcus; the cocci occur singly in pairs and in chains of three or four, and in masses.

On different media after one week:

Bouillon, cloudy, slight, stringy sediment.

Gelatin, growth along the stab, liquifaction along the line of growth is evident, but slow.

Agar agar, diffuse growth on surface of grayish color; distinctly yellow in color in the thicker portions.

Potato, slight, dry, yellow growth.

Litmus milk, marked acid production and coagulation.

LUNG.

Colony 2, "the white colony," shows a diplococcus in all respects morphologically similar to that from the brain.

On media, after seventy-four hours:

Bouillon, slight, clouding, stringy sediment.

Gelatin, growth along stab; no liquifaction.

Agar agar, free growth along the stab; white in color.

Potato, abundant, rather dry, uneven growth; whitish in color.

Litmus milk, no change.

ANIMAL INOCULATION.—December 9th, at 5 P.M., two full-grown healthy house mice were inoculated at the root of the tail with a forty-eight hour blood serum culture of the white colonies, from brain and lung cultures. After inoculation both animals appeared sick, and remained quiet for about twenty-four hours, after which time they seemed active and well. On December 12th the mouse inoculated from the culture from the lung seemed unwell. At 5 P.M. a lameness and spasm of left hind legs were noticed, with inability to use them. At 5.20 animal was found dead.

Post-mortem examination nineteen hours after. At seat of inoculation is a small amount of hemorrhagic exudate; tissues much infected and edematous. Extending down into the left

groin is more extensive infiltration, hemorrhagic in character. Abdominal cavity is free from disease, the organs appear normal, spleen is small; lungs collapsed, normal; heart, right auricle distended with dark blood.

Coverslip preparations from heart's blood, back and groin show a small diplococcus as in the original cultures, lance-shaped, mostly encapsulated, but some are without capsules. The cocci always occur in pairs, end to end. There is also some variation in size.

Cultures were in all respects similar to those of the inoculated organism. The mouse inoculated with the brain culture recovered entirely.

Unfortunately the hospital pathologist was unable to give microscopic results from sections of the brain and cord in time to be included in this paper. I have to thank Dr. Archibald for his untiring energy in watching these cases for me, and Dr. Harold Parsons for the bacteriological notes.

GENERAL SUBCUTANEOUS EMPHYSEMA.

BY A. C. COTTON, A.M., M.D.,

Chicago, Ill.

On March 21, 1900, I was called to see a child in consultation with Dr. Kreuser, who had taken charge of the case that day. The diagnosis of the previous attendant had been "Bright's disease with dropsy."

Patient, Dora C., aged seven years, seven months.

FAMILY HISTORY.—Father, living and well. Mother died at twenty-eight years, of Bright's disease of four years' duration. Closer questioning revealed that the mother had suffered from a cough for several years. Maternal grandparents in good health. Patient was the second child. Her older brother died of "spasmodic croup and bronchitis," death occurring at the end of a series of three general convulsions.

PERSONAL HISTORY.—Patient had varicella, pertussis, mumps, scarlet fever and diphtheria during the first five years of life. None of these diseases developed in severe form and from all she made good recoveries. She was active and apparently in good health until July 4, 1899, when she had an attack of measles, followed by bronchitis. Had never been well and strong since this illness. Cough persisted and she "seemed to take cold easily." In February, 1900, she had a severe attack of bronchitis and during this illness there was a daily afternoon rise of temperature, with profuse night sweats. This last symptom (night sweating) had been present for some weeks but to a less marked degree.

March 16th after a very hard coughing spell, a ridge appeared over the right clavicle. The cough continued paroxysmal and frequent, and the gradual extension of this cushiony ridge in all directions occurred.

There was no history of convulsions or edema. Urine had been scanty and turbid.

PRESENT CONDITION.—Patient, small for age, was held by grandmother in a sitting posture, slightly bending forwards. The face presented a swollen appearance, particularly the lower

lids, which were enormously distended. The skin had a peculiar, waxy color; lips were somewhat cyanotic and the expression was anxious.

Examination of the surface showed great distention about the neck and chest, completely obliterating clavicular depressions, and extending downward over the trunk, especially in the dorso-lumbar region along each side of the spine. Pressure elicited distinct crepitation and left no pitting.

She resisted all efforts to assume any other position than the one mentioned. Her attention was wholly bent upon securing air and repressing the frequently recurring cough. She realized that this increased the dyspnea, and after holding back the cough as long as possible, her hand was silently extended for a glass of water, of which she took but a sip.

Respirations were shallow and rapid (50 per minute); pulse was rapid and barely perceptible; temperature 102° F.

Physical examination by percussion was difficult as she was irritable at being disturbed, and hyperesthetic. However, it was possible to make out hyperresonance over the greater portion of the chest.

From the symptoms the diagnosis of generalized emphysema was made.

During the six subsequent days of life, infiltration of the subcutaneous tissue extended, involving both upper and lower extremities with the exception of palms and soles, the skin becoming tense and shiny.

The paroxysms of coughing increased in frequency, being nearly continuous the last twenty-four hours. There was increasing dyspnea and deepening cyanosis.

The urine showed acid reaction, high specific gravity, no sugar, considerable amount of albumin, with pus cells in great abundance, a few bladder epithelial cells and hyaline casts, besides multitudes of bacteria.

There was no evidence of gastroenteric disturbance. Her appetite became ravenous the last three days.

No *post-mortem* examination or even photographing was allowed nor, in view of the grave prognosis, was radical treatment, such an incision of skin, permitted.

It was subsequently learned that the undertaker reduced the enormous distention by puncture.

But little is said of generalized emphysema in ordinary text-

books. Anderson in the *Twentieth Century Practice* says: "Infiltrations and accumulations of air in the intestinal tissue of the lungs are most frequently seen in young children during an attack of whooping-cough, bronchitis, croup or convulsions or after traumatism, such as tracheotomy. Interlobular emphysema takes place only in young children because as the lung develops the intervals of connective tissue between the lobules disappear. Hence adults do not have interstitial pulmonary emphysema.

"During a violent expiratory paroxysm some of the delicate air cells rupture and air escapes into the connective tissue surrounding the lobules or under the visceral pleura, where it may occasionally be seen *post-mortem* as strings or beads of air bubbles. The amount of emphysema present varies. At times only a few lobules are surrounded by air beads, and again the air may extend along the connective tissue to the roots of the neck, face and general cutaneous surface. Sudden and severe dyspnea after a paroxysm of coughing in a child is strongly symptomatic of interlobular emphysema."

In a hasty review of the literature I have found but few reported cases of generalized emphysema. Gaillard in *Sajou's Cyclopedia*, Vol. v., reports three cases in children of the same family, complicating measles. He thinks there was congenital weakness of the pulmonary vesicles and also a predisposition produced by whooping-cough. Molin, in the *Brooklyn Medical Journal*, October, 1897, reports a case following fractures of the sternum and ribs, in a boy of eight years who was run over by a wagon. The entire subcutaneous area became infiltrated in less than thirty minutes. Within three hours the distention was enormous, and respiration became greatly embarrassed, with marked cyanosis. In this case, immediate relief was secured by incising the integument, the air escaping as from a distended rubber ball. By means of drainage tubes the emphysema gradually disappeared, although after eighteen days some crepitation was present.

A case in a child six and one-half months old, reported by Wrinch in the *Canada Lancet*, January, 1900, gives a history of cough, without, however, severe paroxysms. The child died on the sixth day of the generalized emphysema. The autopsy showed a left pneumothorax, with collapsed lung, which on section revealed miliary tuberculosis. The right lung was honeycombed with cavities. Subsequent investigation proved

the tubercle bacillus present in large numbers in the sputum of the father. The same writer suggests the differentiation of this condition from lesions due to the *bacillus aerogenes capsulatus* by the fact that in the latter emphysema increases *post-mortem.*

Fowler and Godley reported seven cases, several of which followed tracheotomy.

From the histories of recorded cases it would appear that the tendency of extensive general emphysema is towards a fatal termination, either from this condition alone or from the disorder which it complicates. A case came under my observation, as coroner, in which an inquest was held upon a young primipara who died in the first stage of labor, after a fit of violent vomiting, followed within a few hours by general emphysema. The autopsy showed no other cause of death. In this case a torn pleuritic adhesion afforded a point for escape of air. Is it possible that any treatment may ameliorate this condition or lessen the fatal tendency? It occurs to the writer that where the point of entrance of air to the subcutaneous tissue is within reach, as after tracheotomy or external lesions, that the extension of the mischief might be prevented by surgical interference. From the apparent fact that the condition is intensified by coughing or deep respiratory movements, the indications would seem plain to relieve the cough and restrict respiration by any means in our power. Molin's experience with incisions and drainage tubes, and the subsidence of the distention by the undertaker's puncture in my case, certainly would suggest the advisability of efforts to relieve by similar procedures.

ACUTE NEPHRITIS FOLLOWING INFLUENZA.

BY ROWLAND GODFREY FREEMAN, M.D.,

Clinical Lecturer on Pediatrics in the University and Bellevue Hospital Medical College; Attending Physician to the Foundling Hospital and the Seaside Hospital of St. John's Guild, New York.

Among the organic lesions which may complicate influenza one of the rarer is nephritis. A simple albuminuria during the height of a severe attack of influenza is not uncommon. Senator reports having seen albuminuria in 18 of 52 cases. Brandes has seen it in 23 of 27 cases. Teissier in 50 per cent. of cases. Concerning the frequency of the occurrence of albuminuria in children, I have no exact data, but Holt states that most cases of influenza with high temperatures have albuminuria. Krunhals and others have observed hematuria renalis several times.

RÉSUMÉ OF RECORDS OF CASES OF NEPHRITIS COMPLICATING INFLUENZA.

Recent articles on influenza in general medical works, which deal with adults as well as children, make little mention of nephritis complicating influenza. On the other hand, Gmeiner, in a review of about 400 cases, finds that 1 per cent. of his cases had acute nephritis.

Finkler states that nephritis is not a frequent complication of influenza.

In the German Army Reports only 10 cases of severe kidney inflammation were found in 55,263 cases. Lichtenstein had 2 cases of acute hemorrhagic nephritis in 439 cases.

In Bavaria, Finkler states, nephritis seems to be a more common complication, and a number of observers there mention acute nephritis of the hemorrhagic type.

A case with autopsy reported by Leyden occurred in a seamstress, twenty-five years of age, of previous good health.

REPORTED CASES OF NEPHRITIS COMPLICATING INFLUENZA.

No.	Reported by	Age.	Sex.	Duration of Influenza before Nephritis in Days.	Quantity Daily.	Albumin.	Blood.	Casts.	Edema.	Duration of Nephritis in Days.	Termination.	
1	Danforth.	65	Male.	7		Much.	Yes.	Blood and Hyaline.		28	Recovery	
2	Leyden.	25	Female.	35	3 oz.	Much.	"			30	Death.	Autopsy, kidneys large and congested. Glomerular Nephritis.
3	Russell.	Young woman.	"	Early.	Dim.	Yes.	"	Yes.			Recovery	
4	"	"	"	"	"	"	"	Blood.		Few weeks.	"	
5	Mosler.	17	"	28	Dim.						Death.	
6	Fliessinger.	16	Male.			Yes.	"	Epithelial.	No.	8	Recovery	
7	Krunhals.	15	"	8						28	"	Recurrence after 6 weeks which lasted 6 weeks.
8	Sympson.	11	"	16		Yes.	"		Yes.	30	"	
9	Fraser.	9	"	2	Dim.	2%	"		Slight.	26	"	
10	Fliessinger.	8	"	4				Epithelial.	Yes.	14	"	
11	Freeman.	4	"	30	Dim.	Small amount.	"	Blood and Epithelial.	No.	10	"	
12	Fliessinger.	3	Female.	23				Epithelial.	Yes.	17	"	

She became sick with violent headache, slight difficulty in hearing, and since then had never been perfectly well. She became weak and miserable and had anorexia. A month later she had violent vomiting, lasting eight days. Urine scanty and turbid. Edema present. She died at the end of the second month. There was no fever. She passed only about three ounces of urine a day. The urine contained blood and albumin.

Autopsy.—Kidneys large and congested; on microscopic examination a glomerular nephritis of the type found in scarlet fever was present.

Nephritis complicating influenza, although apparently much more common in children than in adults, receives slight attention in pediatric works. Thus, the possibility of the occurrence of nephritis complicating influenza is not mentioned in many recent text-books on diseases of children.

Earle mentions nephritis; Gillet emphasizes it, stating that it may be serious and of the congestive type, as indicated by hematuria; and Holt states that a few examples are on record.

I have been able to find altogether only 17 cases, both of adults and children, of nephritis complicating influenza and concerning only 11 of these cases have I obtained clinical data. These 11 cases I have tabulated with the case I report. This number, 12, is perhaps rather small to form the basis of conclusions, but still this group presents certain interesting features.

It is noteworthy that all but one of the cases, of which I have clinical data for this tabulation, are in children or young adults, and five are in children under twelve years of age, the youngest case having been three years old.

The clinical type of the kidney disturbance, the acute hemorrhagic type, is apparently similar in at least seven of the eleven cases. Seven of these cases occurred in males and five in females. The nephritis may occur early in the course of the influenza or long after the acute stage. Thus of the ten cases in which this date is stated, the second day is the earliest, the thirty-fifth day the latest, the average being the fifteenth day.

The duration of the nephritis, which is definitely stated in nine cases, was but eight days in the shortest case and thirty days in the longest cases, giving an average of nineteen days' duration.

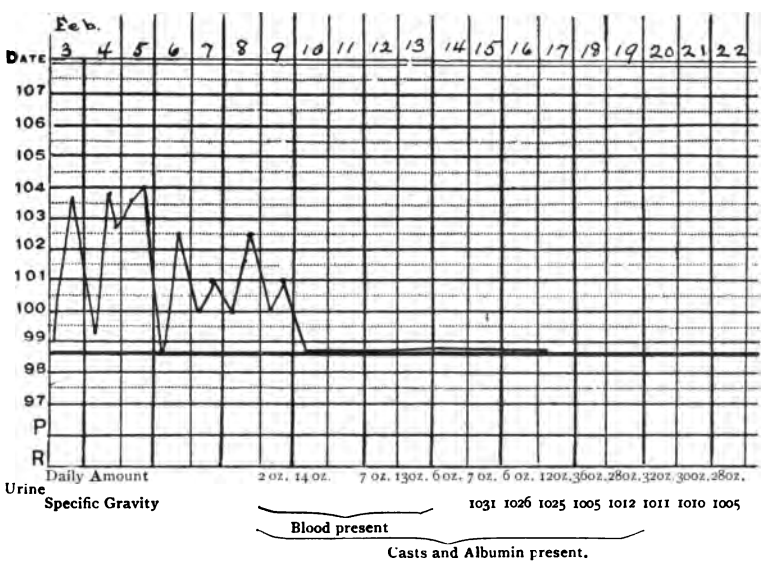
The daily quantity of the urine was in several cases remarkably diminished, albumin was present in varying amounts, and

blood and casts of different sorts are in most cases noted as having been present. Two of these cases showed no edema while a third showed a slight edema.

The prognosis in nephritis complicating influenza as indicated by the cases I have selected is good, ten of them having recovered and only two having died.

Although the pathological change in the kidney is reported in only one of the tabulated cases, I have records of the examination of 6 cases.

Of these 4 show parenchymatous degeneration; 1 shows parenchymatous degeneration, with fatty degeneration; 1 shows glomerular nephritis.



TEMPERATURE CHART AND PARTIAL RECORD OF EXAMINATIONS OF THE URINE IN A CASE OF INFLUENZA WITH COMPLICATING NEPHRITIS.

My own case is a boy, four years of age, who has for the past three years suffered each winter from an attack of influenza. The present attack began about January 1, 1899, with the ordinary catarrhal symptoms, prostration, fever, and moderate attacks of earache of a few hours' duration, but at no time was there any discharge from the ear. On January 31st he had a temperature of 102.5° F., the highest temperature being 105° on February 5th. The temperature gradually diminished, making an irregular curve, so that by February 9th it

varied between 100° and 101°. On this day the child passed some very red urine. The urine had previously been examined on February 4th, and then had a specific gravity of 1022 and contained no albumin and no blood. On the 6th the urine was noticed to be red, but was not examined. On the 7th and 8th it was pale. The urine of February 9th was passed in very small amount, only two ounces being recorded on the chart, and contained a considerable amount of blood, about 5 per cent. by bulk of albumin, and casts, both hyaline and containing blood cells. On the following day, the 10th, Dr. W. P. Northrup saw the child with me and made an unqualified good prognosis. The temperature on this day reached normal and remained constantly normal afterward, but the urine continued to be passed in small amounts, varying from six to fourteen ounces a day. Blood remained present for five days and the casts for ten days. At the end of this period the casts and albumin disappeared and the urine was excreted in the normal daily amount of about thirty ounces. There was absolutely no edema. The child did well and has had no recurrence of albuminuria during the following year.

An interesting phenomenon took place two months later, after a slight disorder of the bowels, with diarrhea, when the child passed no urine during one night, and the following morning passed about half an ounce, which was very red and contained considerable deposit. This urine had a specific gravity of 1050, contained no albumin, casts or blood, but abundant urates and phosphates.

This child, then, had an acute nephritis of the form which, judging from the data at hand, appears to be the most common clinical type in the nephritis complicating influenza and made a good recovery.

Conclusions based on a very limited number of cases:

1. Although albuminuria is fairly frequent with influenza, nephritis is a rare complication.

2. The nephritis complicating influenza is clinically of the acute hemorrhagic type and morphologically shows toxic lesions.

3. It apparently attacks children more often than adults.

4. The kidney disturbance may appear a few days after the acute symptoms of the influenza, or as long as a month later.

5. The prognosis is good.

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DISCUSSION.

DR. FRUITNIGHT.—Nephritis is very rare as a complication of influenza. I have a record of 57 recent cases of influenza. I found a trace of albumin in only 1 case, the urine had a specific gravity of 1012, the quantity was less than fourteen ounces. In a few days the albumin had disappeared. I attributed the albuminuria to the febrile condition, just as we see albuminuria so often in simple febrile conditions. In 57 cases, if it were at all common, more cases of nephritis should and would have occurred, and in this only instance in which albumin was present I would consider the case one of transitory albuminuria rather than one of nephritis.

DR. DORNING.—Nephritis seems to be a rare complication of influenza, and I think Dr. Freeman's presentation of the subject will stimulate us to examine more carefully our cases of influenza in children. I have had 3 cases of acute nephritis complicating influenza in children, 2 of them in the same family. In 2 cases no attention was given to the urine until one child showed some edema of the face. The urine showed about 30 per cent. of albumin and a large number of blood casts and epithelial casts. It was for three weeks before the casts and albumin entirely disappeared. Another child, four years old, showed edema of the face some two weeks later. In that child's urine was also found some epithelial casts and some blood casts. In a few days the urine cleared and the child seemed perfectly well. In another family a boy of four years showed a very pronounced form of nephritis. After I had discontinued my visits to the family the mother brought the little fellow to my office stating that his face had been swollen for a few days but the swelling had disappeared. The day before she brought him to the office the swelling had reappeared and extended over the whole body, including the scrotum and pre-

puce. I found epithelial casts, blood casts and some hyaline casts, and about 50 per cent. of albumin. The little fellow complained of headache and loss of appetite, and these were all the symptoms. This case occurred six weeks ago, and the urine has not cleared yet. The edema disappeared in some six days, but still the urine contains casts and about 5 per cent. of albumin. His treatment has been by baths and laxatives, and a milk diet with just enough solid food to break the monotony of the milk diet. He had plenty of water to drink.

DR. JENNINGS.—In my observation nephritis is a very rare complication of influenza, and but 1 case, and that now under observation, has come to my experience. This is a case of nephritis following a double infection, influenza appearing first, and after an interval of three or four days of convalescence an attack of measles, and then after three or four days of convalescence the nephritis appeared. I believe nephritis is also rare in measles, and it is difficult to determine which infection had most to do with the attack. The child was six years old. At the first observation the amount of urine was sixteen ounces in twenty-four hours, specific gravity 1015. An abundant precipitate of albumin and an abundant deposit of hyaline and blood casts and epithelial *debris* were the urinary findings. The urine cleared in a very short time; the blood and albumin disappearing on the third day. The granular casts persisted in few numbers for, perhaps, a week. The urine increased to twenty ounces in twenty-four hours on the third day, and to twenty-eight ounces on the fifth day, and after that time the child was convalescent. There were only two days during which any temperature was noted. The second day the temperature was 102° in the afternoon, on the third 100°. Aside from this febrile movement, there were none of the ordinary phenomena of nephritis. It was an exceedingly mild attack.

DR. ROTCH —There is scarcely any disease in children in which the pathological conditions in the organs affected correspond so indefinitely to the clinical symptoms. It is almost impossible to make a diagnosis of any special form of renal disease in young children by means of an examination of the urine. That is, if you follow the rules which you would in making the diagnosis in adult cases. Often, for instance, we find clinical symptoms corresponding to glomerular nephritis where the pathological conditions found after death do not correspond. It is of very great importance that we should all endeavor to make notes on the pathological conditions and try to get some other symptomatology for making a diagnosis. At least this has been our experience in Boston, and the same is true, from what I can gather from the reports in other cities. The most important question is as to the form of nephritis. The acute interstitial nephritis running an acute course just as glomerular nephritis does is more characteristic in young chil-

dren than it is in adults. An acute interstitial nephritis found *post-mortem* is usually secondary to some infectious disease, such as diphtheria or measles, while glomerular nephritis is more characteristic of scarlet fever in young children. It would be interesting if we could see some autopsies following influenza. It is probable the nephritis after influenza is of the interstitial type. The last case spoken of by Dr. Dorning seemed to me to be one probably of acute interstitial nephritis, although we do not know enough about these cases yet to make a really definite statement. In like manner Dr. Jennings' case may have been an acute interstitial nephritis, although the amount of albumin would correspond more to glomerular nephritis. So far as I know a sufficient number of *post-mortem* examinations have not yet been made in these cases, and I believe that they can be determined only by *post-mortem* examinations, as it is difficult to differentiate between the acute interstitial and glomerular nephritis following infectious diseases in children. As to nephritis following influenza in children, I have seen a large number of cases of influenza, and I find that nephritis does not occur often; I therefore agree with the gentleman that it must be a rare complication. I would treat such cases as we do our cases of scarlet fever and measles, being very careful about the diet, and not taxing the kidneys.

DR. DORNING.—I would like to ask Dr. Rotch's experience in interstitial nephritis in children regarding the quantity of albumin in the urine. I have not had much experience along that line in children. In the adult we know it is not uncommon in interstitial nephritis to find a small percentage of albumin or none at all. I do not know whether Dr. Rotch lays stress on the amount of albumin as indicating an interstitial process, a glomerular involvement or an acute parenchymatous inflammation.

DR. ROTCH.—I do not think we can judge as in adults as to that. The interstitial nephritis resembles in its clinical symptoms very much the glomerular nephritis. I do not think we know enough about it yet. We have not advanced sufficiently far in our study of the condition in children. The *post-mortem* examinations do not give pathological findings corresponding to the clinical symptoms and to the examination of the urine.

DR. CARR.—A case very similar to that reported by Dr. Freeman came under my care this past winter, the only difference was perhaps that the catarrhal symptoms in the nasopharynx were followed by suppuration and perforation of the tympanum. The clinical phenomena were very much the same as those narrated. The examinations of the urine gave a specific gravity that varied between 1017 and 1022 for some time, and the urine showed a small amount of blood, blood casts and epithelial casts. What struck me most was the very small amount of albumin. At no time was it more than a trace. The child

had a limited amount of edema of the face, feet and hands. Under dietetic management and a certain amount of medication the child improved. But one of the striking things in the case was that the child suddenly developed a liking for sugar. The specific gravity of the urine went up from 1024 to 1040; the appearance of the urine did not change, and it was not dark or smoky. There was no increase in the albumin, but sugar was shown by the fermentation test. The urine improved and at the last examination, made about ten days ago, there was a specific gravity of 1007, without albumin, casts or anything pathologic. The interest in the case was to decide whether I was dealing with a child who from any cause might have had a previous attack of nephritis. I could find absolutely no trace in the urine of anything that looked like a hyaline cast or a cast of a pre-existing disease, and there was no evidence in the child's circulatory apparatus that there had been any such thing. I thought it was more than likely that there was an interstitial change, but I am inclined to reserve that opinion with a view to further developments. The case was of added interest because of the desire for sugar, the sugar in the urine and the limited quantity of albumin. I have had 49 or 50 cases of influenza in children in which I have examined the urine without finding albumin or any evidence of nephritis.

DR. CHURCHILL.—The subject of nephritis in influenza is one of the greatest importance and one to which, by the great majority of physicians, not nearly enough attention is paid. Even if physicians examine the urine at all, they examine only for albumin; and if they do not find albumin they are satisfied there is no nephritis present. My own experience is that actual nephritis in influenza is rare, but I would like to raise the question whether these cases are not often the starting point of serious renal trouble developing later along in life, either at puberty or even still later, and that we may often find trouble by more careful and exhaustive examination of the urine. We will find what you may call simply renal irritation, without possibly an actual nephritis. In a great many cases of influenza and other infectious diseases an examination of the urine shows what are known as cylindroids and not hyaline casts. These persist for months after the acute symptoms subside, although we find no albumin and the child is apparently in perfect health. These cylindroids have been regarded as evidence of renal irritation and not of actual nephritis. I have a very interesting case in a youth of nineteen, to whom I was called for an attack of appendicitis. I examined the urine, although there were no symptoms of kidney trouble, and I found a nephritis. The boy had had an attack of influenza eighteen months before, at which time the urine was not examined. He had measles at seven years, and since infancy he had had a great deal of chronic intestinal trouble. The question arose whether the nephritis had its starting point in the measles and was con-

stantly aggravated by the intestinal trouble, or in a possibly slight irritation at the time of the influenza eighteen months before I saw him. Of course, there is no way of determining this question. I think it is an extremely important point to examine the urine in all these cases to see if there be the slightest evidence whatsoever of irritation, and if we find such irritation they should be very carefully followed up for months or years until we see the disappearance of such characteristics. I should like Dr. Freeman, in closing, to state in regard to the sudden increase in the amount of urine, what the diet of the child had been before, and if any change was made at that particular time. And then, I did not understand whether he said the specific gravity was 1050 or 1015. If it was 1050, was there any sugar present?

DR. CAILLÉ.—From a clinical standpoint I make a diagnosis of nephritis when I find albumin and blood casts. Taking these as diagnostic points, I believe nephritis is rare in influenza. There is still another important point in Dr. Freeman's paper, and that is his statement that the prognosis is usually good. I have seen the most aggravated cases of nephritis in children clear up completely, and I have for twenty years had under observation individuals, who in their earlier years had nephritis and have since remained absolutely well. Their urine has been examined frequently, and they have remained absolutely well as far as the kidney is concerned. It is important to bear in mind that the most desperate cases of nephritis in children may clear up completely, and the prognosis, therefore, is not necessarily unfavorable in children.

DR. ROTCH.—I would like to add my experience to that of Dr. Caillé. The most desperate cases may get well in children. I would like to ask Dr. Freeman how much the temperature depended on the ear, and whether it is the temperature chart of nephritis or of otitis plus nephritis. I think that should be clearly stated so as not to be misleading.

DR. FREEMAN.—In reply to Dr. Churchill's question I will say the specific gravity of the urine was 1050, and there was no sugar. There have been no nephritic symptoms during the year that have elapsed since this attack. In regard to the ear as a possible etiological factor in this case, there was no evidence of otitis except the child's occasional complaint of earache. During the month preceding this attack he had at times pain in the ear, which would pass off in a few hours; but there was no otorrhea or any tenderness of the ear or any evidence of inflammation on examination with a speculum. I felt some hesitation in presenting this small number of cases and drawing any conclusions from them, still they are the result of a rather exhaustive search of the literature of the last ten years; and I have hoped that by drawing attention to this matter some other cases might be reported and we might learn more about this condition.

CONGENITAL CARDIAC MALFORMATION WITH ENDOCARDITIS AND ANURIA.

BY A. C. COTTON, M.A., M.D.,
Chicago, Ill.

Baby G., born March 19, 1900. Family history negative. Mother, a primapara, had been in good health during gestation. She had never suffered from rheumatism. Labor was normal. Weight at birth, seven pounds four ounces; length, twenty-two and a half inches. A well-developed child, presenting no external malformations. Meconium was passed shortly after birth. Although respiration was not delayed, it was noted that the skin did not assume the usual deep red color. There was a marked pallor which persisted, changing to a grayish hue, actual cyanosis supervened gradually as a late symptom.

On the second day the temperature rose to 102.8° F.; pulse, 160; respiration, 48. Examination showed a loud, harsh, diastolic cardiac murmur which was heard all over the chest. It was impossible to locate the exact position of this murmur.

No urine was voided, and catheterization showed an empty bladder. Lactation was established on the third day. The child nursing ineffectually, the breasts were pumped and the infant fed from a tube. There was no vomiting nor any evidence of gastrointestinal trouble.

The baby was not restless nor irritable, crying only when disturbed. At no time were there any symptoms of eclampsia. The breathing steadily increased in rapidity, became labored and of a marked abdominal type. Anorexia became more pronounced, with a steady failure in muscular vigor. The child died of progressive asthenia on the fifth day, no urine having been secreted.

AUTOPSY BY PROF. HEKTOEN.—Infant G., aged five days. A well-developed, well-nourished female; the body still warm; the umbilical cord is dry and there is a line of separation around its insertion at the navel; rigor is strong; the surface of the body is livid.

The serous cavities of the trunk are empty, their lining smooth. The diaphragm reaches to the fifth rib.

The pharynx, larynx and trachea are normal. The thyroid and thymus are normal. The lungs are distended and contain many areas of hemorrhage, subpleural and deep-seated. The lungs crepitate freely; no bronchitis; much congestion.

There are subepicardial extravasations at the base of the heart. The heart is distended with blood. There is great enlargement of the heart, especially of the left ventricle. From

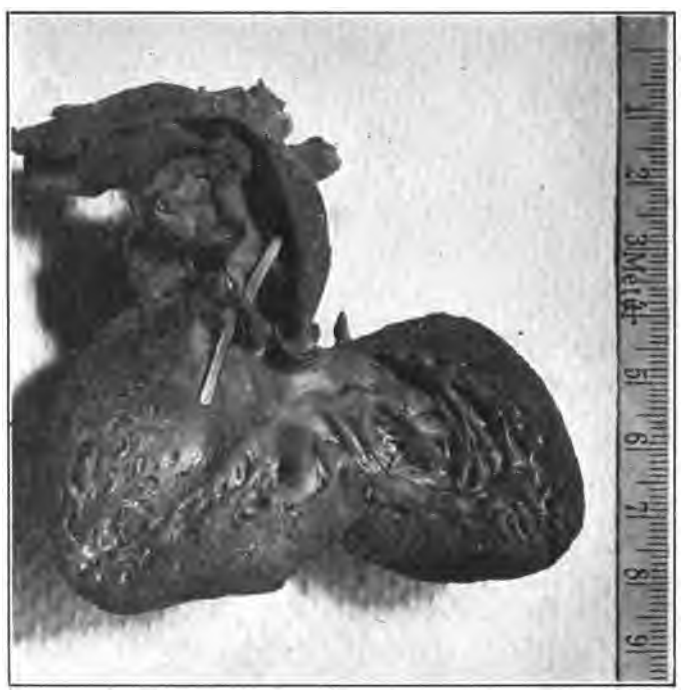

Fig. I. STICK PASSED THROUGH OPENING BEHIND ANTERIOR SEGMENT OF AORTIC VALVE.

base to apex the heart is 4.5 cm. long; 5 cm. across widest base. The heart weighs 42 gms. The endocardium of the right side is normal. The depth is 4 cm., the wall is 3 mm. thick. The tricuspid orifice is 1 cm. in diameter. The foramen ovale is patent and the ductus arteriosus is large and widely open; the aortic orifice measures 6 mm. across.

The pulmonary artery is normal. The mitral orifice is small—about 5 mm. in diameter; the auricular surface of the valve

is smooth but nodular, and there are delicate but smooth outgrowths and nodules upon the cordæ tendinæ. No ulceration, no thrombosis. The aortic valves are large, higher than normal (about 6 mm.), irregularly thickened, but smooth, there being small, delicate, reddish nodules upon them so that they appear somewhat deformed. There are three valves, but the anterior segment is attached to an irregular band-like bridge, 2 mm. thick, (see Fig. I.) which extends across an oval depression in the upper part of the interventricular septum and the lower aspect of the aorta which here presents a marked bulging outward between aorta and pulmonary artery. It looks as if the lower end of the aorta and the interventricular septum had failed to meet fully. The depression is 1½ cm. vertically, 1 cm. across and 8 mm. deep. The bulging is smooth and there is no communication between it and the pulmonary artery or the right ventricle. The interventricular septum is not perforate. The left ventricle is 4 cm. deep and the wall is 5 mm. in average thickness. The beginning of the aorta just above the sinuses of valsalva is wider than usual, but smooth; it is 2.5 cm. in circumference; coronary openings are normal; coronary arteries are normal.

The liver is large, congested, bluish in color, and weighs 127 grams. It is smooth.

The spleen is congested and weighs 7 grams.

The kidneys show marked uric acid infarctions in the medullary pyramids. The ureters and bladder are normal. The brain could not be examined.

DIAGNOSIS.—Chronic endocarditis of aortic and mitral valves (mitral stenosis); defect at base of aorta (aortic regurgitation); hypertrophy of heart; general passive congestion; dilated ductus arteriosus.

POINTS OF ESPECIAL INTEREST.—1. The left sided congenital lesion (Rauchfuss, of St. Petersburg, finds, out of 300 cases of fetal endocarditis, only 15 showing left sided lesion alone); 2. Unusual character of the malformation (the writer has thus far failed to find any record of a similar case); 3. Negative maternal history as to rheumatism, infectious or specific diseases; 4. Absence of early cyanosis with such a manifest lesion.

QUESTION OF INTEREST.—What is the relation of congenital malformation of the heart to anuria? The blocking of the kidneys by uric acid crystals forcibly recalls the case of anuria with congenital cardiac malformation presented by the writer at the

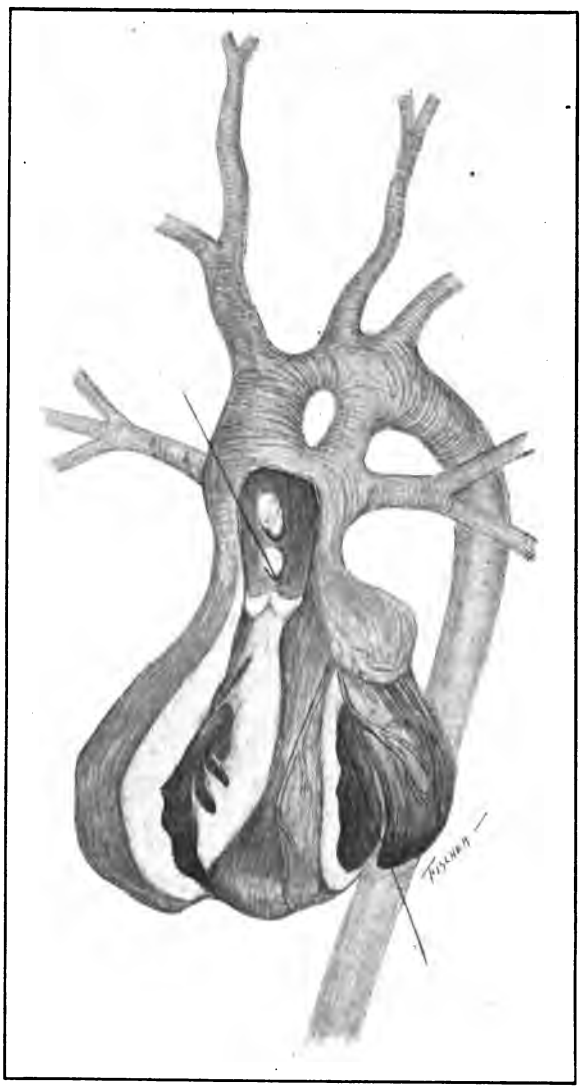

Fig. II. COMMON TRUNK FOR AORTIC AND PULMONARY VESSELS. "ARCHIVES OF PEDIATRICS," OCTOBER, 1899.

last meeting of the American Pediatric Society (ARCHIVES OF PEDIATRICS, October, 1899).

In that case the heart showed a common trunk for aortic and pulmonary vessels, as shown in the drawing. (Fig. II.) At least two other cases have come under my observation, in which death from anuria occurred the first week of life in babies showing heart anomalies. The exact nature of these anomalies could not be ascertained, autopsies being refused.

If we might accept the older theory that uric acid precipitation be due to suboxidation, then we might reason that any cardiac malformation which interfered with the circulation of oxygenated blood through the kidneys would favor the accumulation of uric acid in these structures. Late authorities seem inclined to discard this Vierordt theory, in favor of the one of which Horbaczewski is a recognized expounder. This is that uric acid is derived in the body from the disintegration of proteids containing nuclein, notably from the leucocytes. Accepting this theory of the derivation of uric acid from the nuclei of leucocytes affords a rational explanation of the frequency of infarcts in the new-born, a marked leucocytosis at that age being acknowledged. It has been shown that diet which favors leucocytosis also increases deposition of uric acid. The question as to whether the latter be derived from the nuclein of proteid ingested or from the nuclein of the leucocytes called out by digestion, matters little, it seems to the writer, so long as it is admitted that thorough oxidation is necessary for complete metabolism, with resultant urea.

The well-known frequency of uric acid infarcts at a time when the heart is physiologically incapable of furnishing arterial blood to the renal arteries, on account of the still patent foramen ovale and ductus arteriosus further strengthens this opinion. The writer sees an analogy between this condition and the congenital cardiac deformities, which are practically a persistence and aggravation of the conditions which normally obtain in the first few days of life.

1485 JACKSON BOULEVARD.

DISCUSSION.

DR. ADAMS.—Did the child have any diarrhea?

DR. COTTON.—No; some calomel was administered and the bowels responded.

DR. ADAMS.—How many movements did the child have in the twenty-four hours?

DR. COTTON.—The first normal meconium was passed in the first few hours, and after that there were one or two movements a day in response to small doses of calomel.

DR. ADAMS.—I do not know whether Dr. Cotton places this case as a septic endocarditis or not. To what do you attribute the elevation of temperature?

DR. COTTON.—There is a question mark. For instance, there are some who believe that bacteria may produce a septic endocarditis, and the toxins may produce a smooth endocarditis. We had here the little plastic nodules.

DR. ADAMS.—I have recently ound reported two cases of congenital septic endocarditis. These cases were found really by collateral reading rather than the reports of cases as such. The subheads are often quite confusing. In reference to the question of the relation between the cardiac lesion as described and the anuria, it does not seem to me that one is responsible for the other. We frequently find, in septic endocarditis, infarctions, not uric acid infarctions always, but usually septic infarctions. It would be interesting to have had a bacteriologic examination. I have one case under observation now of septic endocarditis, not in an infant however. There is no remission in the temperature except from the hydrotherapy. The septic cases have a septic temperature. This baby whose case is reported lived five days with complete anuria.

DR. BLACKADER.—Had the mother suffered from any rheumatic affections?

DR. ADAMS.—That part of the history was entirely negative.

DR. COTTON.—I would say in regard to the temperature, there must always be a question mark in the etiology of temperatures. Dr. Holt has put himself on record concerning an inanition temperature in the first few days of life. There have been a number of confirmations of that as a possible cause of temperature. Here we had conditions extremely favorable for the development of an inanition temperature, if we may recognize that as an etiological factor. The child nursed poorly and was fed with pumped milk through a tube, and somewhat ineffectually. The text-books treat anuria too lightly. There have come under my observation a number of cases, I can think now of 6 fatal ones, in which there was anuria. Of these 4 showed anomalous heart conditions. The 2 others, in which no autopsy was allowed, were recognized as blue babies with evident abnormal heart murmurs. It seems to me that it is certainly suggestive of some relation between the cardiac organic difficulty, which actually interferes with the oxygenation of the blood, and the uric acid infarct. So far as a bacteriologic examination is concerned, the report is not complete, because I have not heard from the pathologist. Some slides are being made, and I will be only too glad to report on them.

ATRESIA OF THE LARYNX DUE TO TRAUMATISM, THE RESULT OF FAULTY INTUBATION.

BY W. P. NORTHRUP, M.D.,

New York.

There is a certain amount of sentiment connected with the presentation of this paper and its accompanying specimen. It was the last case of intubation seen by the late Dr. O'Dwyer, and as you will remember it illustrates the subject of the paper last presented by him before this Society.*

This is a specimen of atresia of the larynx, apparently the result of faulty intubation, *i.e.*, caused by faulty technique. Looking backwards, it seems probable that it was a case of subglottic stenosis from the first.

Dr. O'Dwyer in his last paper, referred to above, says: "The cause of persistent stenosis following intubation in laryngeal diphtheria can be summed up in the single word—traumatism." He next asserts that it may be due to (1) faulty tubes; it may be due to (2) faulty technique, *i.e.*, it may be the fault of the intubator. The tubes used in this case were of the latest pattern, approved by Dr. O'Dwyer himself; the technique was that of a beginner and proved faulty. Here, then, is a case of traumatism resulting in cicatricial contraction and finally complete stenosis of the larynx.

A. B., ten months, was sick at first with "bowel trouble," so the physician in charge asserted. The child gradually became hoarse and developed the following symptoms: hoarseness, croupy cough, croupy inspiration, croupy expiration, dyspnea, aphonia, cyanosis. The pharynx was red and swollen; no exudate was seen at any time. Intubation was demanded. The operation was performed by a young friend whom I had instructed, and who rehearsed the operation upon the cadaver sufficiently, I thought, to justify him in doing it upon his case. He inserted the tube fairly well, leaving the string attached. I assisted the operator and was inclined to congratulate him upon having

* "Retained Intubation Tubes—Causes and Treatment." By J. O'Dwyer, M.D. Transactions of the American Pediatric Society. Vol. ix. 1897.

done well for a beginner. Further instruction was given concerning feeding and general care of intubated patients, and the case was left in the care of the physician and a trained nurse seemingly in a satisfactory condition. The subsequent history was that there was need of repeated (three) intubations, two extubations and an accident which necessitated tracheotomy. The second and third intubations were required because the child got its hands free and dragging out the tube so suddenly that the nurse, though watchful and careful, was taken entirely off her guard. Extubation is always more difficult than intubation, and it is probable that this was the occasion of greatest harm to this patient's larynx. Five intubations and two extubations, in the hands of a beginner, reveal ample cause for the lesion here shown.

Two things constantly worked against the success of intubation and gave Dr. O'Dwyer great uneasiness of mind: viz.: imperfect tubes and imperfect technique. Each brought reproach upon the operation. Each meant traumatism to the larynx; each worked to the harm of the patient; each diminished the chances of recovery. The inventor wrote always in the same strain, endeavored to keep it before the minds of the medical profession that the operation should be done by skilled hands and should include proper tubes.

I am permitted to show you this specimen for the sake of the lesson conveyed. We learn from our accidents; this specimen tells its own story; faulty technique has its monument in the specimen which is here shown.

The specimen shows scar tissue extending across the calibre of the larynx at the level of the cricoid ring. It is nearly like the constriction of an hour-glass in its middle. A probe passed from above is arrested in a cul-de-sac; passed from above, the probe catches in a similar cul-de-sac. The closure is complete; no air could possibly have passed. An accident rendered tracheotomy necessary. Through the operation wound the endeavor was made subsequently to open the channel through the larynx, but obviously without success. That is a matter of surgery and apart from our present purpose: viz., to point the lesson of faulty technique in intubation. It has meant in this case retained intubation tube, laceration of larynx, finally atresia. The child's death was due to pneumonia.

The patient was seen often by Dr. O'Dwyer just before he was taken sick, and often during his illness he discussed the prognosis and treatment. The treatment, you will be interested to recall, was outlined in his last paper and consisted of inserting a hard rubber tube and leaving it in place some weeks to allow the ulcerated larynx to heal. The hard rubber tubes, you may recall, were not yet on the market, but the manufacturer, Ermold, had promised them for the near future and a sample Dr. O'Dwyer showed on that occasion. Such a tube this child wore for some time until the accident which necessitated the tracheotomy.

DISCUSSION.

DR. MILLER.—I think Dr. Northrup is rather severe on the operator. I have seen somewhat similar cases occur in the practice of very skilful men, and while I believe that intubation is an operation every practitioner should be able to perform, I think, where we can get the service of one who is skilled, it ought always to be done. Unless a man is very familiar with the technique he will find it, often, a difficult operation.

DR. COTTON.—It would be very interesting to know in how many cases some previous lesion or possible traumatism may have affected the calibre of the larynx. This occurs to me as I recall a case in my own practice some thirteen years ago in which I was called by the physician in a hurry to relieve a child that had intense dyspnea with a diagnosis of diphtheria. I attempted to introduce a tube to relieve the dyspnea. The conditions were not good, and no assistance was at hand except the young physician who was with me. To make a long story short there was a failure to relieve the dyspnea, no instruments being at hand for the opening of the trachea, and the child succumbed. I was very anxious to know why I failed. That larynx removed from the body showed a stenosis from a previous traumatism. There was an apparent cul-de-sac posteriorly in which the point of the tube caught every time. I am very sorry that the specimen has been lost, and I have no photograph of it.

DR. NORTHRUP.—As to a cul-de-sac excited by previous traumatism, that is quite impossible in this case, for the child then would never have breathed. We used O'Dwyer's tubes, but the operation was obviously faulty. The moral is, not

everybody can do an intubation safely. You notice this is a case of subglottic stenosis, and that means a swelling of the membrane within a restricted ring; that is, it swells to the detriment of the calibre, the ring being resistant. That pressure made the hole through which the intubation tube was to go naturally smaller, and putting it in four or five times took all the membrane off, bringing the submembranous tissue to the surface and made it favorable for stenosis. Practically it is a case of traumatism. But if we want to say honestly what is the fact in these cases it is, not everybody can do intubation without detriment to the patient.

COUGH IN INFLUENZA SIMULATING WHOOPING-COUGH.

(PSEUDO PERTUSSIS, PERTUSSOID).

BY F. FORCHHEIMER, M.D.,

Cincinnati.

The first difficulty arrived at is to find a proper name for the combination of symptoms which forms the basis of this paper. Great care must be taken in introducing new names, both on account of the fact that the nomenclature of disease is overburdened, and also because a new name more or less necessarily means a new disease. For this reason, principally, I have refused accepting the term, pseudo pertussis, introduced by the Italians, and pertussoid, used here and there in German literature, although both or either describe the clinical condition thoroughly well. Beyond the first objection urged to new names, none can be applied to the name pseudo pertussis. The name pertussoid is a barbarism, combining as it evidently does a Latin name with a Greek ending, and if for no other reason should be discarded.

The paper is confined to the combination of symptoms to be described, in connection with influenza, although these symptoms, more or less developed, are found in other conditions besides influenza and whooping-cough proper. It may be well, before going further, to state what I mean by influenza. In looking through the literature on this subject I find that various authors mean various things. There can be no doubt of the fact that the diagnosis, influenza, has been grossly abused since 1890. I have seen the most remarkable things attributed to influenza. On the other hand, there is a tendency to reduce the diagnosis of influenza to limits that are not justifiable.

Von Jaksch insists upon making the diagnosis of influenza only when the influenza bacillus of Pfeiffer is found. Leichtenstern makes the statement that the absolute diagnosis of influenza can only be made when this bacillus is present, but afterwards modifies his statement by saying that the diagnosis can rest upon the finding of the influenza bacillus, unless at some future time it may be shown that some other bacillus produces influenza as well. At the same time, however, he is willing to admit that the diagnosis of influenza can be made upon clinical evidences. We owe to him possibly the differentiation between influenza vera and influenza nostras, the difference between the two being represented by the difference between Asiatic cholera and cholera nostras, influenza vera always being produced by the bacillus of Pfeiffer. I do not hesitate to say that the diagnosis of influenza cannot be limited to the finding of the bacillus of Pfeiffer, and that for several reasons. In the first place, this bacillus had not been unconditionally accepted as the cause of influenza. The requisites of Koch have not been fulfilled. Secondly, in a disease that occurs as a pandemic, it would be manifestly impossible in order to make the diagnosis to examine every case for the influenza bacillus. Lastly, it has been recently shown by Hueppe, that in the last few epidemics of influenza it has been almost impossible to find the influenza bacillus after the first few days of the disease. All these cases then, if we depended solely upon the finding of the bacillus for our diagnosis would have to be rejected as not being influenza. I prefer to base the diagnosis for the present upon clinical evidences, and in this respect am more inclined to agree with Filatow, who claims that besides the ordinary symptoms, influenza vera in children is characterized, first, by the fact that it is pandemic, secondly, that it is not confined to place nor restricted by season, and thirdly, that it attacks all human beings, young and old alike. To this there may be added several other things; that clinically it is confined to certain types, which can be well grouped under three headings, the gastrointestinal, the respiratory and the nervous, to which might be added possibly a fourth, the hemorrhagic type; that one attack does not produce immunity but on the contrary a predisposition, and that influenza is characterized by certain sequelæ and complications which are not found in connection with the ordinary grippe or influenza nostras. For the cases that form the basis of this paper these

requisites have been fulfilled. While I hunted assiduously and carefully for the presence of the influenza bacillus, it must be confessed that with the exception of three or four cases the influenza bacillus was not found, in all of them, however, streptococcus. The bacteriological examinations were made after all precautions required for the examination of the influenza bacillus had been taken. I have also borne in mind the fact that Doernberger states that in 45 per cent. of healthy children streptococci were found in the mouth. It is true that 14 out of his 40 children had carious teeth. Possibly this may account to some extent for the difference in his statement and those of Netter, 5.54 per cent. and Kurth, 4.5 to 8 per cent. For this reason as well as to make the determination an accurate one, material for examination was always taken from *within* the expectorated material. As far as the bacteriology then of this condition is concerned, the evidence of disease being the result of true influenza is absent. In every case that was examined we found streptococci. It was impossible to make a positive diagnosis of the kind of streptococcus present. Animal experiment upon white mice showed that they were not pathogenic. But if the criticisms of Von Jaksch were to be applied, he having reported cases similar in some respects to the cases that we will have to deal with, these cases must be ascribed to the streptococcus infection and not to influenza. Notwithstanding this, on account of the definition that I have given of influenza, I do not hesitate to state that these cases were true cases of influenza vera.

The symptoms in these cases at first were very puzzling and the differential diagnosis between true whooping-cough and influenza whooping-cough in the beginning, and I might add throughout the observation of this peculiar form of trouble, was very difficult in individual cases. In the first place, note must be made of the fact that this complication is not a new one, having been described as far back as 1510 by Short; and in the next place, it must be remembered that the French name *coqueluche*, before Baillou described whooping-cough in 1578, was the name that was given to influenza in France, and then applied to whooping-cough. In 1892, the Italian, Guidi, described pseudo pertussis. He was followed by Pestalozza and Musatti in 1893, who in the main agreed with the statements made by Guidi. Filatow, 1892, describes a condition like the one we are discussing, but does not bring it under the head of

a disease, very similar to whooping-cough. In the discussion on influenza at the Hot Springs Meeting of the American Pediatric Association, 1895, Holt and Blackader mentioned a cough simulating whooping-cough, and Leichtenstern, in his excellent monograph on influenza, 1899, makes mention of the act of the existence of whooping-cough-like cough.

The peculiarities of this cough are as follows: It always moved in epidemics; it was decidedly contagious. If it broke out in a family, few or none of the family were spared, irrespective of age. The servants who came in contact with the patients were attacked, and in three instances I have seen patients attacked who had previously had whooping-cough, to which a further reference will be made. The onset was that of an ordinary attack of influenza. In children there was fever and principally the form of the respiratory or gastrointestinal type of influenza. These symptoms would pass over possibly in two to four days, and then would begin a cough. If the type had been originally the respiratory form, the cough would immediately develop into the characteristic cough, which shall be described below. If the original attack had been in the form of the gastrointestinal type, all the symptoms would have disappeared and then the peculiar cough would develop. This cough is characterized by the following: It usually first develops at night, but not so distinctively as in whooping-cough, as the attacks would appear during the day time as well. The cough is that of whooping-cough except that the peculiar whoop is not so characteristic as in pertussis; however, sufficiently so to be recognized as a whoop, and in some instances as well marked as we find it at any time in whooping-cough. The cough is accompanied by the ordinary congestive symptoms of whooping-cough, is followed by vomiting and expectoration, in every respect like whooping-cough. The mouth in some respects differs from whooping-cough in that the peculiar blue color of the mucous membrane and of the tongue are absent. This is to be ascribed to the fact that the attacks of coughing are more numerous but not so long in duration as those of whooping-cough. As to the ulcer of the frenulum, my observation has shown that it not only is present, but if anything better marked than in whooping-cough. And in this respect as well as in some others, I differ from Pestalozza and Musatti, who have not seen this ulcer in what they call pseudo pertussis. Not only was the ulcer well developed, but on

account of the frequency of the attacks in a large percentage of the cases there were additional ulcers on the tongue, due to the protrusion of the tongue against the lateral incisors.

In this form of trouble as well as in whooping-cough implication of the bronchial tree varies with the individuals and the epidemic. I have had occasion to study this form of trouble in four epidemics and in each one complications on the part of the bronchial tree varied. Upon the whole, I think that I am justified in coming to the conclusion that in those cases in which the original attack of influenza was respiratory, the chances of a bronchitis or pneumonia developing were greater than in those cases in which the original attack was confined to the gastro-intestinal tract. The duration of the trouble varies very much, depending largely upon individual attack, but also upon the treatment. If left to itself the disease lasts as long as six to eight weeks, and longer, but in the majority of instances it can be aborted so as to last not longer than from a week to ten days. Pestalozza refers to the fact that children who have once had whooping-cough may have similar attacks for years afterward, and seems to offer this as an explanation for the appearance of this cough. I have been singularly fortunate in being able to observe a family of children who had never had whooping-cough, but who became affected with this whooping-cough-like cough, and after they had recovered were infected with true whooping-cough, which was brought to them by a visitor from another part of the country.

The only explanation that seems plausible to me is the one that in order to produce symptoms of whooping-cough there must be a certain anatomical location of the cause, and this cause need not of necessity be the specific cause of whooping-cough, whatever that may be, but any one localizing itself in or upon a certain part of the respiratory apparatus.

The complications and sequelæ are largely those of whooping-cough. Some, however, must be ascribed to influenza. In another place, Jacobi's Festschrift, I have described the complications on the part of the heart. In four cases I saw develop typical influenza pneumonia. In a large number of cases there was present croup; in one case subglottic croup, which required intubation and tracheotomy; in several cases in adults symptoms of edema of the glottis. In three cases there was developed meningitis, and in five so-called cryptogenetic septicemia, the origin of the septicemia being a streptococcus bronchitis.

As has been stated, the differential diagnosis was difficult at times, but in the majority of cases it could be made when the principal points of difference between this form of disease and whooping-cough were borne in mind, the epidemic appearance of this form of trouble, associated with a general epidemic of influenza; the fact that the disease attacked adults as well as children, former attacks of whooping-cough not protecting against this form of trouble. As far as I myself am concerned, my observation is in accord with Vogel-Biedert, Eichhorst and Henoch, who have never seen a second attack of whooping-cough in the same individual, Theodor, on the other hand, having seen it fifteen times in 353 cases. Then comes the peculiar relation of the cough to the original attack of influenza, the influenza following its usual course, whatever that may be, in the individual case, followed thereafter by this peculiar cough, the peculiarity of the cough again being that while it increases in severity, unless there be a catarrhal cough present, within twenty-four to thirty-six hours of the onset it is fully developed as a whooping-cough-like attack.

Having made reference to treatment as a factor in the duration of the disease, brief mention of the method that I have employed may not be amiss. In the presence of a house epidemic of this form of trouble, or even when this form of trouble is epidemic I have found that *full* doses of quinia materially reduce the duration of the disease. But the dose must be sufficiently large, not less than one decigram less than the age in years and one centigram less than the age in months. The quinia cuts the disease short, only when it is given in full doses and given early; it has been my rule to give these large doses of quinia as soon as I suspect the development of this peculiar cough. When the cough has developed thoroughly, phenacetin or antipyrin gives great relief, but has very little effect upon the duration of the cough. In very bad cases codein must be given in full doses and sometimes it becomes necessary to give chloral at night in order to insure sleep. In most cases belladonna is a disappointment, but in some, especially when the cough has persisted for some time, its effects are very gratifying. When symptoms of septicemia have developed, and mild symptoms are not infrequent, unguentum Credé or even the injection of streptococcus serum has proved of great benefit in my experience.

EPIDEMIC PARALYSIS IN CHILDREN.

BY HENRY DWIGHT CHAPIN, M.D.,

New York.

The occurrence of epidemics of paralysis in children has been reported in recent years by a number of observers. They have generally been considered as cases of anterior poliomyelitis, and have naturally provoked renewed discussion as to the essential cause of this disease. The prevailing idea among recent writers appears to be that the spinal paralysis of children is an infectious disease, and occasional epidemics confirm this view. Thus, Church and Peterson (1899) state that its infectious nature is indicated by the abrupt onset, the usual febrile movement, the gastric disturbance, the occasional occurrence of convulsions, and most of all by epidemic and endemic outbreaks. Such have been recorded by Colmer, Cordier, Medin, Leegard, Oxholm, Nonne, Calverly, Altman and others. The authors find in some of these outbreaks that a considerable variation from the type has been noticed, and some cases presented the symptom complex of Landry's paralysis, the infectious nature of which is known. It must be borne in mind, however, that while the microbic origin of poliomyelitis, may, by analogy, be assumed, it has not been scientifically demonstrated. Gowers' recent volume (1899) in discussing this subject states that we must remember the vascular activity that all function entails, and the readiness with which the vasomotor system of children is disturbed. The occurrence of the disease in more than one member of the same family indicates a congenital disposition of the system to react in a similar manner to certain external agencies, while a special feature in the latter may perhaps explain the occasional epidemic character of the disease, and furnish grounds for ascribing to it an infective character. The evidence available suggests that it is probably due to some chemical change in the blood analogous to that which seems to cause rheumatic fever, though probably distinct from it —a change excited by cold, disposed to by the effect of heat,

the result of some derangement of metabolism which we cannot yet understand, and perhaps having underlying these effects some organismal cause. Doubtless there is more than one possible cause for poliomyelitis, sometimes of an infectious nature, sometimes due to refrigeration and again to a possible traumatism. Dr. Sachs relates the cases of two children in the same family being attacked with spinal paralysis and within two weeks of one another. Careful inquiry showed that the disease in both cases came on shortly after a very cold surf bath. It is fair to suppose, however, that in epidemics there is an infectious element present. This is further predicated by the fact that in these epidemics the paralysis may not be exclusively spinal, as other parts may be affected by the supposed microbe. Dr. W. Pasteur (Clinical Society's Transactions, Vol. xxx., 1897) reports an epidemic of infantile paralysis occurring in the seven children of one family. Three were followed by permanent paralysis, two had a primary fever without paralysis, one had a primary fever followed by general tremors lasting a few days, and one had primary fever followed by partial tremors lasting a few days and strabismus of short duration. He concludes that there was a unity of cause in this group of cases and thinks they furnish a conclusive proof that a poison which in one case gives rise to a typical anterior poliomyelitis can determine in others lesions in other parts of the nervous system. It is highly probable that these phenomena will be seen in all epidemics of paralysis in children. An interesting epidemic occurred during the summer of 1899 at Poughkeepsie, N. Y., most of the cases being attacked between the middle of July and the middle of August. Seven of the cases were seen by me in consultation with Dr. D. M. Sheedy. The peculiarity of this epidemic appeared to be the existence of severe pain in the parts affected by the paralysis.

The following case is fairly typical:

George C., aged four years. Never been sick. Illness started with vomiting, high fever and pain in the limbs. First seen on July 20th with temperature 104° F., and pulse 120. He complained of severe pain in back and limbs. July 21st, temperature 102° F., pulse 100. There was quite an arch in his back, giving an appearance of tetanus. On the fourth day after the onset his temperature was normal, the pain had pretty well subsided and he had lost control of his limbs. An examination in the follow-

ing February showed that he was able to stand up by a chair and walk around it. There was also considerable wasting of his legs. The cases examined by me with Dr. Sheedy showed absolute paralysis of the limbs affected, with loss of reflexes, and apparently considerable pain on handling the part. There was such marked evidence of the action of some infectious principle that I suggested a blood examination, and accordingly specimens were sent to Dr. H. T. Brooks, who returned the following report: "Microscopic examination of the three blood slides sent to me by Dr. D. M. Sheedy, of Poughkeepsie, at your suggestion, failed to give any positive results. The specimens did show occasional minute microorganisms (a diplococcus) to which, however, I cannot attach any etiological significance, because of the small number of specimens and also because the latter may have been contaminated from the skin or other source. It was also noted that the so-called hematoblasts of Hayem or blood plaques of Bizzozero were somewhat increased above the normal, which finding, according to recent researches, is said to be indicative of dissolution of red elements, the number of plaques being in direct proportion to the degree of dissolution. As such a condition could also be present in any state attended by reduction of the red elements by dissolution or fragmentation (if recent views are correct), I would hesitate to attribute any great significance to this phenomenon. I believe that reliable data can be secured in the cases under observation only by a large series of complete blood examinations, and by histological and bacteriological examination of the body tissues and fluids other than blood." During this epidemic the general health of Poughkeepsie was unusually good, according to Dr. Sheedy, and there did not seem to be evidence of malarial infection in the locality. The prominent feature of pain in these cases, and its more or less persistence in the affected limbs, brought up the question of neuritis. One of the cases proving fatal, an autopsy was made, and the nature of the disease in this particular instance proven.

John S., aged two years, died in convulsions twenty-four hours after he was taken sick. Dr. Brooks removed the cord for examination and made the following report: "The microscopic examination of the cord structures did not show anything more than has already been well described in many standard works on pathology, *e.g.*, Ziegler's 'Lehrbuch der

Speciellen Pathologischen Anatomie'; Orth, Langerhan, Israel, etc., namely, hemorrhage, affection of the ganglion cells of the anterior horns, characterized by cloudy swelling, shrinking and swelling, granular and hyaline degeneration, vacuolation and disintegration. In those specimens stained for bacteria, however, there was observed a diplococcus morphologically resembling the microorganism referred to by me in my former report to you in reference to the blood specimens. These cocci were found in the degenerated area, *i.e.*, in the anterior horn region, but not in the peripheral or meningeal portion of the cord. This appeared to me to be of striking interest; still I would hesitate to draw any etiological conclusion therefrom, because of the possible sources of error which may at any time arise during the course of such examinations, and which cannot always be avoided. However, considering the clinical histories of such cases, the acute character of the disease, the epidemic form in which it sometimes manifests itself, and the localization of the pathological changes, the results are, to say the least, quite suggestive."

In reports received from Dr. Sheedy during February and March, he tells me that the great majority of the cases observed by him made a complete recovery in from one to four months. Three cases, however, had paralysis and atrophy, with the typical after appearance of poliomyelitis. A little girl of seven years, whom I saw in the acute stage, with pain and paralysis of the lower extremities, was examined by me in April, when I failed to find any loss of power in the parts previously affected. It is certainly very rare to find complete recovery after an attack of acute anterior poliomyelitis. I never remember to have seen such a case. In this epidemic, the prominence of pain with the paralysis, followed by complete recovery in many of the cases, leads me to consider these as attacks of peripheral neuritis. With the data at hand, I believe we can conclude that this epidemic was of an infectious nature, that in some cases the infective principle attacked the anterior horns of the spinal cord, in others, the peripheral nerves, and that possibly, in a few cases, both parts were attacked.

51 WEST FIFTY-FIRST STREET.

DISCUSSION.

DR. CHAPIN.—I have never seen a marked case of poliomyelitis permanently and entirely recover. But a large percentage of the children in this epidemic have completely recovered, and from that, I believe we can state that the disease was limited to the periphery. Other epidemics have been noted. Dr. G. M. Hammond tells me they had one at Bridgeport some years ago, in which the paralysis was in some cases spinal and in some cases peripheral. I believe at times the disease is limited to the cord, in some to the peripheral nerves, and possibly in a third it will be found in both.

DR. ADAMS.—Was any lumbar puncture made?

DR. CHAPIN.—No.

DR. GRIFFITH.—It was my privilege a year ago to see an instance of the occurrence of poliomyelitis in two members of one family, two children being taken ill within a few days of each other, without any cause that we could discover. This has tended to strengthen my belief that the disease is an infectious one.

It might be of interest to state that those of you who may look up the published report of the epidemic of neuritis to which Dr. Chapin has referred, will find there the account of one very typical case of infantile scurvy with its pseudo-paralysis.

DR. CHAPIN.—I will simply guarantee that none of these cases was scurvy.

DR. GRIFFITH.—I did not mean to infer that any of Dr. Chapin's cases were instances of scurvy, as I am sure he would not make this mistake in diagnosis.

MALARIAL COMA IN CHILDREN.

BY GEO. N. ACKER, M.D.,

Washington, D. C.

During the past year I have had under observation two cases of coma connected with malarial fever, which I thought were caused by the malarial parasite.

On looking up the literature on the subject I was surprised to find how little had been written about it. It is true that in pernicious malarial fevers the congestive and comatose varieties have been described—in some cases preceded by convulsions.

Dr. H. B. Anderson in the *Peoria Medical Monthly*, December, 1883, gives the history of three cases of "Malarial Fever in Children With Meningeal and Cerebral Complications." When these cases are analyzed two are found to be due to abscess of the middle ear, and there is some doubt as to whether malaria was the real factor in the causation of the symptoms as no examination of the blood was made.

Since the systematic study of the blood in every case of fever in hospitals is now made by competent men, and the different stages of the malarial organism is so well known, cases which were formerly treated under some other name are now being classified correctly. Thus we find that malaria is responsible for many conditions such as congestion of the lungs and kidneys which were not attributed to it.

It is remarkable that malaria in children does not cause more disturbance of the nervous system than is usually the case, for with the unstable condition that is found in poorly nourished children one would expect the cerebrospinal system to be chiefly affected resulting in paroxysms characterized by delirium, convulsions, coma and tetanic spasms producing serious disturbance of the function of animal life.

CASE I.—I. C., aged eleven years, male, colored, was sent to the Children's Hospital, August 31, 1899, by the Health Department with the history that the child had been unconscious for three days. No other history could be obtained.

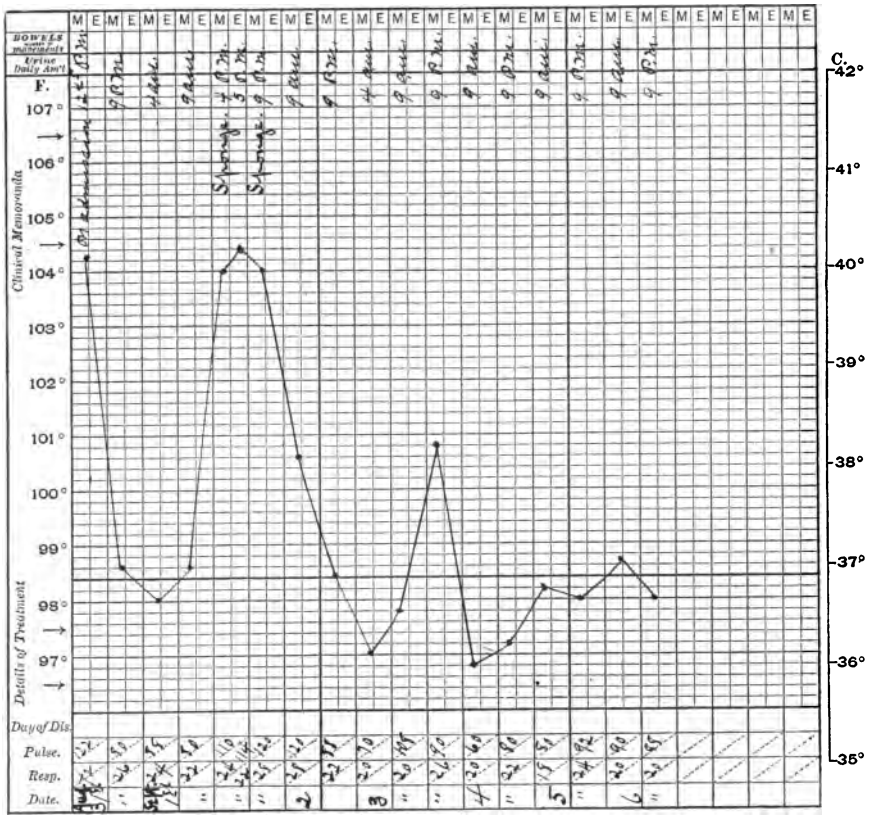

CHART I.—CASE OF ISAAC CARTER, 11 YEARS OLD.

Admitted August 31, 1899.

PRESENT CONDITION.—The child is in a comatose condition, with tonic spasms, and cannot be aroused, lies on back with forearms flexed on arms and the legs cannot be extended when the thighs are at right angles to the body. The eyes are staring and the mouth is tightly closed. Pulse 122 and respirations 42 per minute. Lumbar puncture was done at 4.30 P. M. and though no fluid came away, yet in an hour the patient began to speak and take nourishment and at the same time the muscular spasms relaxed.

September 1st.—Slept well during the night and appeared rational. During the day slept a great deal, and passed a large quantity of urine which on examination was found normal. An

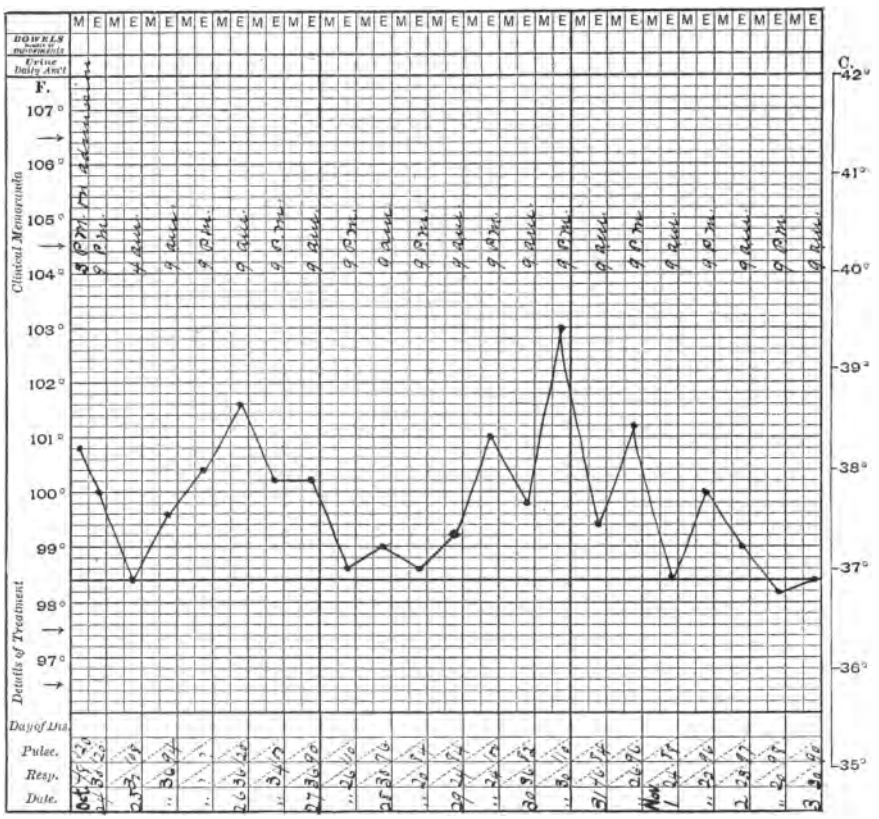

CHART II.—CASE OF B. R., 8 YEARS OLD.
Admitted October 24, 1899.

examination of the blood showed a large number of the estivo autumnal organism. Five grains of the muriate of quinin was ordered every three hours. The temperature rose to 104.5° to-day.

September 2d.—Slept quietly during the night. He had a chill between 12 and 1 P. M. The limbs were rigid, eyes set and he appeared in a state of coma for two hours. Perspired freely at 4 P. M.

September 3d.—Has a good appetite and seems much improved. The patient had a slight rise in temperature to-day followed by a profuse perspiration.

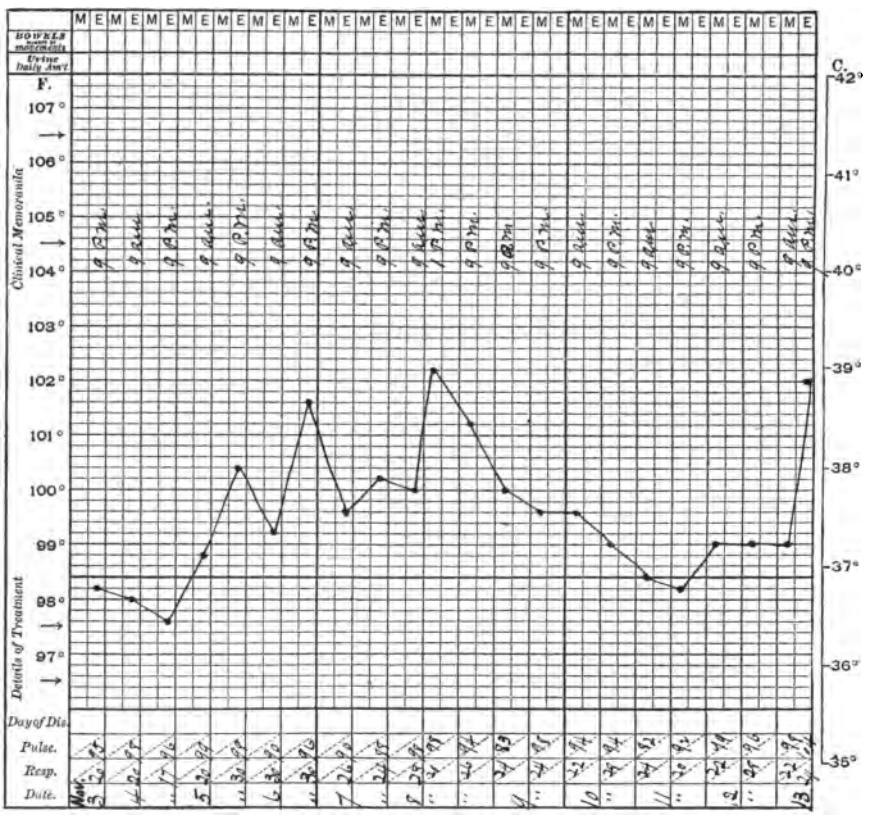

CHART III.—CASE OF B. R. (*Continued.*)

September 5th.—He had some slight rigidity to-day. The urine has been examined several times with negative results.

Small doses of quinin muriate and Fowler's solution were continued for some time and he was discharged cured October 3d. (Chart I.)

CASE II.—B. R., female, colored, aged eight years, was admitted to my service at the Children's Hospital, October 24, 1899.

FAMILY HISTORY.—Father (white) living and in good health. Mother died three years ago with pneumonia. No other children. No syphilitic or tubercular history obtainable.

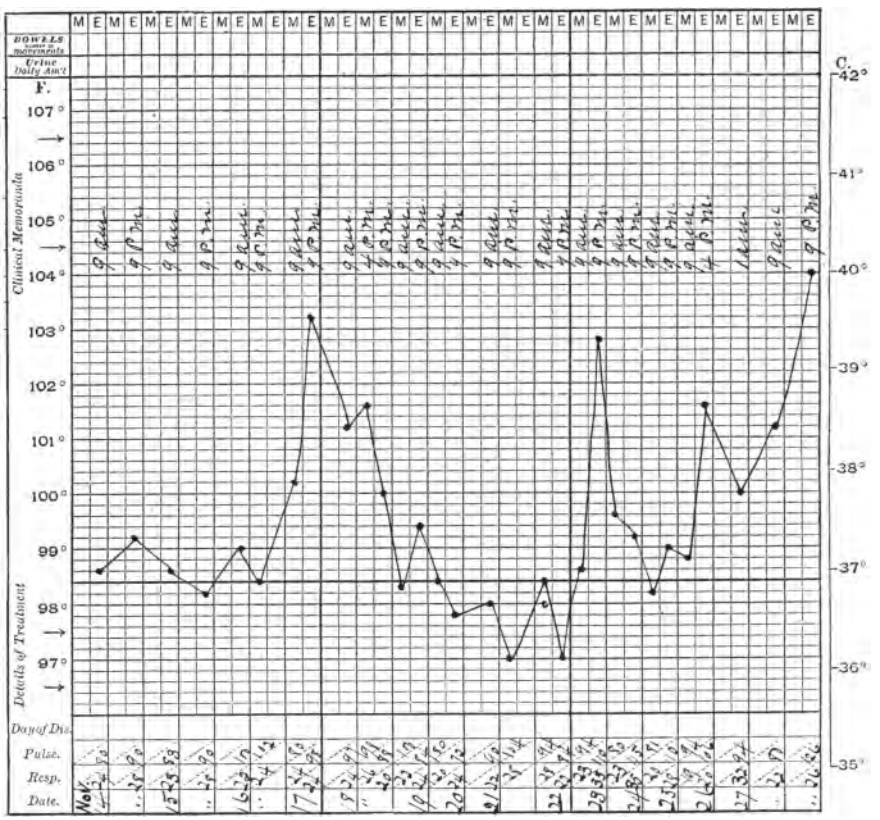

CHART IV.—CASE OF B. R. (*Continued.*)

PREVIOUS HISTORY.—Labor was long and tedious and the child was born asphyxiated. She was fed at first on condensed milk three teaspoonsful to the pint and half of water and about the end of the first year was put on cow's milk. Began dentition under one year of age and progressed normally. When one year of age had cholera infantum. Present illness began three weeks ago with coughing spells and high fever coming on at night. This was followed by swelling under the eyes and of the extremities, accompanied by great shortness of breath, especially at night. The fever has continued and she has been restless at night. She had two severe convulsions just before being admitted to the hospital.

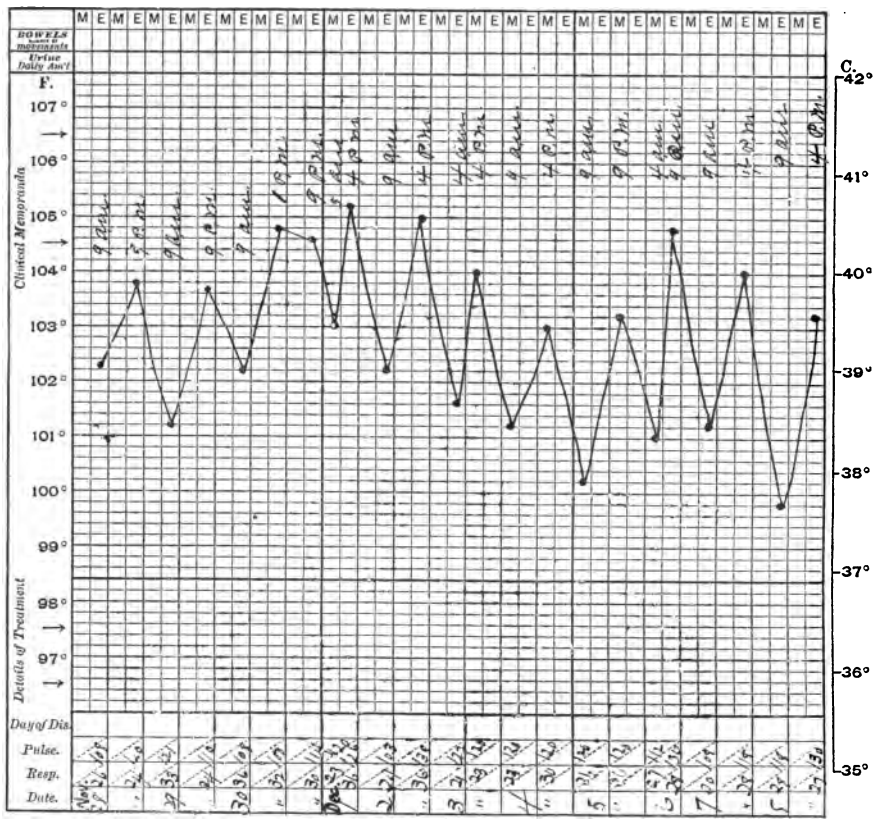

CHART V.—CASE OF B. R. (*Continued.*)

PRESENT CONDITION.—The child is well nourished. Lies on back as if exhausted. The skin is light in color and without eruption. There is some swelling of the feet. The cervical lymph nodes on the left side of the neck are slightly enlarged.

The respirations are 42 per minute, deep, and without pain. She has a slight cough. There is some rough breathing over both lungs but no râles. The pulse is 92 per minute, regular and weak when first admitted, but became slower and stronger in a few hours.

The appetite is poor and bowels are regular. The tongue is heavily coated and breath offensive. The liver and spleen are normal. The child has pains over the eyes and in abdomen.

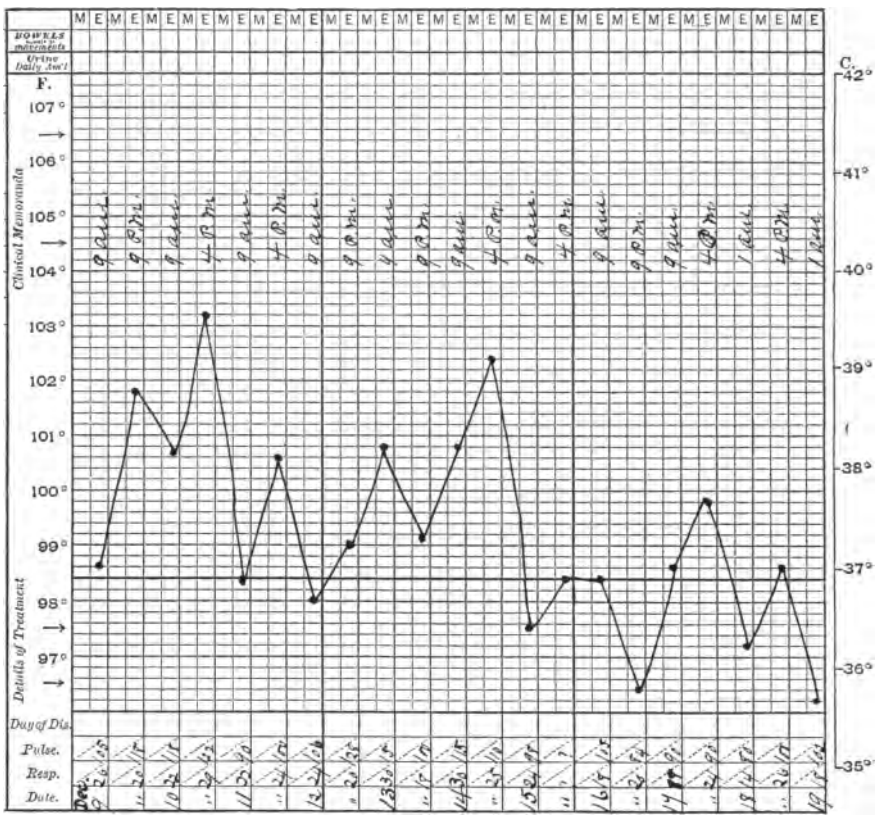

CHART VI.—CASE OF B. R. (*Continued.*)

It was necessary to catheterize her twice during the day. The urine was of a pale straw color with heavy sediment. Reaction acid, specific gravity 1020, urea eight grains to the ounce, albumin 4 per cent. Few epithelial, granular and hyaline casts with few blood corpuscles and many leucocytes.

October 25th.—Slept and took nourishment well during the night. Swelling about the feet less and child brighter. Temperature was normal at 4 P.M. About 7 P.M she had severe convulsions, clonic in character, lasting about ten minutes. The skin was hot and dry; she was placed in a steam tent and given twenty grains of bromid of soda and ten grains of chloral hydrate by the rectum. This attack left her nauseated. She

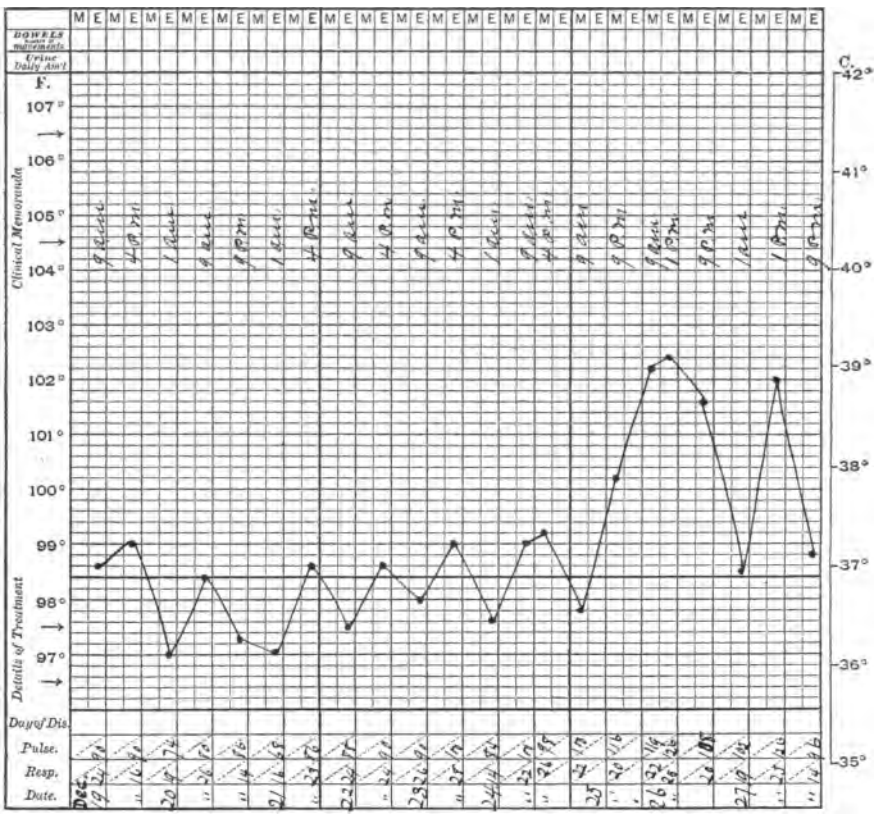

CHART VII.—CASE OF B. R. (*Continued.*)

had slight convulsions at 9 P.M., and at 10.15 P.M. another very severe one occurred which lasted fifteen minutes. The convulsions appeared to affect all the muscles. She voided thirty-one ounces of urine during the day.

October 26th.—She was unconscious all night. Became conscious at 8 A.M., but at 9.15 A.M. was restless with muscular twitchings followed by hard convulsions lasting ten minutes. She remained in an unconscious condition until 1.15 P.M. when she had a convulsion of half an hour's duration. Inhalations of chloroform kept her quiet all the afternoon. The hot applications were continued, but the skin did not act until a hypodermic of pilocarpin was given when she perspired freely. There were two loose stools from elaterium. She passed urine invol-

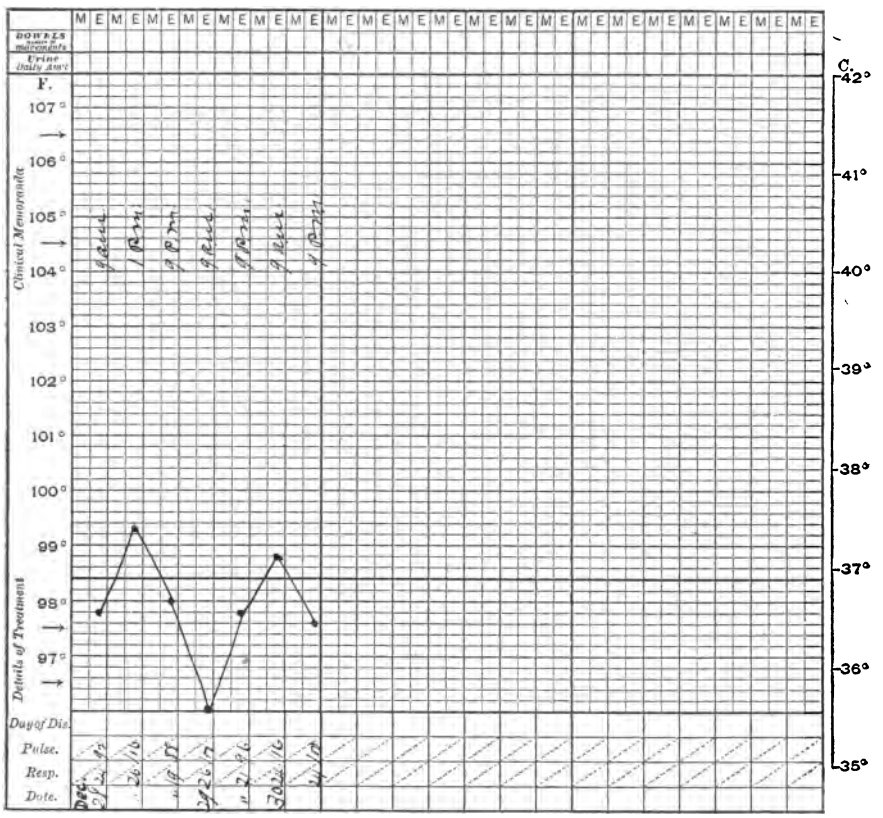

CHART VIII.—CASE OF B. R. (*Continued.*)

untarily so that the amount could not be ascertained. Albumin 3 per cent., no casts, few blood corpuscles with some leucocytes. On the examination of the blood numerous estivo autumnal organisms were present.

October 27th.—Slept quietly the greater part of the night. Took nourishment well and asked frequently for water. Large stool from enema. Pulse slow and regular. Is dull, but appears conscious. Was nervous at times during the day. Quinin muriate grs. 5 and strychnin sulph. grs. 1-120 were given every three hours. About twenty-seven ounces of urine passed in twenty-four hours which on examination showed 3 per cent. of albumin, few blood corpuscles and some leucocytes. Seven grains of urea to the ounce.

October 30th.—Rested well the last three nights. The girl is brighter. She has had several large stools daily from compound jalap powder. There has been about twenty-five ounces of urine voided daily. The analysis to-day was as follows: Amber color, acid reaction, specific gravity 1015, eight grains of urea to ounce, ½ per cent. of albumin; few red blood corpuscles and many leucocytes.

November 5th.—General condition good. About the same quantity and character of urine with a mere trace of albumin.

November 10th.—The child had been in a good state. The urine has increased in quantity. There is only a trace of albumin with few leucocytes. Methylene blue was given to-day in place of quinin.

November 11th.—Had a slight convulsion to-day lasting five minutes. (Charts II.-III.)

November 14th.—The patient remains about the same. Passes thirty-five ounces of urine of a blue color. Analysis about the same.

November 17th.—The child's temperature took a sudden rise to 103.2° F. and she complained of pain in the epigastrium. Since the 15th inst. she had 3 grains of muriate of quinin every three hours, and though the methylene blue was stopped then there is a trace of it in the urine.

November 23d.—The child had chilly feelings followed by rise of temperature to 102.8° F. and complained of pain in the epigastrium. The urine is normal.

November 27th.—The pains in abdomen have continued and have been marked on the right side.

As the chart shows the temperature increased and ran a remittent course until December 15th when it became subnormal. On the 25th it took a sharp rise with rapid pulse and respiration and slight headache. After running an intermittent course for a few days it became normal. She improved rapidly and was discharged cured January 15, 1900. This attack was proved to be typhoid fever by the number of rose spots which appeared December 5th and a positive Widal reaction. There was some albumin in the urine from the 7th to the 9th of December. (Charts IV.-VIII.)

Without doubt in the first case the coma was caused by the influence of the malarial parasite upon the nervous system. An interesting feature in this case is the effect the lumbar puncture

had upon the comatose state. In regard to the second case there is some question on account of the nephritis which complicated it—as to whether the convulsions and coma were directly produced by the malarial organism. It will be remembered that the active stage of the nephritis had occurred before she was admitted to the hospital and after that time the urine improved in quantity and quality. It is reasonable to take the view that both the kidney disease and nervous disturbances were caused by the malarial parasite.

913 SIXTEENTH STREET.

POISONING BY VAPO-CRESOLENE.

BY S. S. ADAMS, M.D.,

Washington, D. C.

These cases are reported because vapo-cresolene is to be found in a great many houses in which there is a child with a cough. It is sometimes introduced and recommended by the physician. I have seen two cases of carbolic acid poisoning directly attributable to the inhalation of the fumes from a vapo-cresolene lamp. In one case I was called to see a patient who was said to be dying and the family and physician did not know what was the matter. I found the child, aged one year, in coma, and in a cold, clammy sweat. There was marked pulmonary edema. When I asked what had been done with carbolic acid, I was told that the child had been shut up for twenty-four hours in a small room inhaling the fumes from a vapo-cresolene lamp. I asked the mother: "How long has this child been passing black urine?" and she said it had passed black urine, but had passed no urine for twenty-four hours. The child was taken out in the open air, given water to drink and it recovered.

I was called to see an infant aged six months dying, it was said, from pneumonia. The child had stridulous respiration, mucous râles over both lungs, a cold, clammy sweat, and dilated pupils. The temperature was only a little over one hundred degrees in the rectum, and had been even lower. As I went out of the room I saw a vapo-cresolene lamp burning. Somebody had recommended using the vapo-cresolene lamp and the mother had put it beside the crib at bed-time. At twelve o'clock the child refused its food. At four o'clock the mother was awakened by a peculiar noise the child was making, and it was after this that I was called. This patient did not pass smoky urine. The child was taken into another room, and given plenty of water. The odor of carbolic acid was very

perceptible. Usually physicians have attributed no harm to the vapo-cresolene lamp, but I ask for your experiences. Whether the pulmonary edema was due to the congestion of the kidneys or not I am unable to say. This second case also recovered. The pulse and temperature soon became normal and the physician in attendance then said he thought it was a case of "suffocative catarrh." But I do not think there is any doubt about the diagnosis of poisoning by vapo-cresolene.

1 DUPONT CIRCLE.

HEMORRHAGE INTO THE SUPRARENAL CAPSULE IN STILL-BORN CHILDREN AND INFANTS; REPORT OF A CASE SHOWING RUPTURE OF THE SAC AND ESCAPE OF BLOOD INTO THE PERIRENAL TISSUES AND THE PERITONEAL CAVITY.

BY S. McC. HAMILL,

Instructor in Clinical Medicine in the University of Pennsylvania; Pediatrist to the Howard Hospital and to the St. Christopher's Hospital for Children, Philadelphia.

Congestion of the various viscera in the new-born is common. This is especially true of the abdominal viscera. The extensive experiences of Mattei[1] and Spencer[2] in making autopsies on still-born children and infants led them to believe that some degree of congestion of the suprarenal gland is practically always present in the newly born. Macroscopically the border line between slight interstitial hemorrhage and marked congestion is narrow, and where a microscopic examination is not made it is probable that congestion is frequently mistaken for hemorrhage. Spencer, recognizing this fact, defined his cases as follows: As cases of congestion those in which the medulla of the organ assumes a deep red or brownish-red color encroaching upon the cortex, and as hemorrhage those in which there are distinct hemorrhagic spots in the tissue of the organ, or where the walls are separated by a wide line of deep black color, or where the organ is converted into a sac containing liquid or clotted blood. Normally, in the new-born, the gland should show on section a narrow yellowish-gray cortex and a reddish-brown medulla. Between the opposed surfaces of the medulla

NOTE.—Through the kindness of Dr. Richard C. Norris I was privileged to make the autopsy on this case and to report it.

there is a fine red line. This line is much more marked and the medulla of a deeper brown color in the new-born than in adults or older children. The dark red rounded spot in the intermedullary line, marking the central vein, is also more distinct, and unless one bears in mind the presence of this vein and the fact that several such veins may sometimes exist, it is possible from the macroscopic appearance alone to mistake the normal condition for one of limited hemorrhage. Before entering further into the study of the subject I desire to present a complete report of one case and to make brief reference to two other cases which have come under my notice.

CASE I.—The mother of the infant was a healthy woman, aged twenty-seven years. She denied syphilis and showed no evidences of the disease. This was her first pregnancy, the labor was normal, easy and not prolonged. Her husband had deserted her, and she was unable to give any information regarding his antecedent or present condition. The infant, a male child, was born at 8 P. M., on the 6th of January, 1900. He seemed perfectly normal at birth and so continued until the evening of the second day, when suddenly he became pale and his temperature was found to be 103° F. His fever continued until his death. He performed his functions normally and took his nourishment well. He had a slight reddish papular eruption over the upper chest and back, and over the flexor surfaces of the arms. He cried for several hours following the onset of the pallor as if in pain. He became cyanosed and his respirations rapid and labored several hours before death, which occurred suddenly at 1 A.M., on January the 10th.

The autopsy was made about twelve hours later. There was some bluish *post-mortem* discoloration in dependent portions, and some yellowness of the skin and conjunctivæ. Rigor mortis was marked. The cord looked healthy. On opening the abdomen blood exuded, and about six (6) ounces of dark-colored, partially clotted blood was found in the peritoneal cavity. The clotting was especially marked in the right upper quadrant along the under surface of the liver. The umbilical vein was free. The liver extended about one and one-half inches below the xyphoid cartilage and one inch below the margin of the ribs in the right mid-clavicular line. There was some congestion of the omentum and mesentery. On pushing aside the intestines there was found in the region of the right

kidney a mass the size of a duck's egg and having the appearance of a kidney very much distended with blood. It measured 8 centimeters in length by 4 centimeters in width. It adhered slightly to the under surface of the liver. The portions of the ascending and transverse colon which passed over the mass were closely adherent to it. The sac ruptured during its removal, with the escape of a considerable quantity of dark colored and partially clotted blood.

On section the mass proved to be a hemorrhage into the right suprarenal capsule, with rupture into the perirenal tissue almost completely surrounding the kidney. The latter was

CASE I.—EXTENSIVE HEMORRHAGE INTO THE RIGHT SUPRARENAL GLAND AND PERIRENAL TISSUES COMPRESSING THE KIDNEY. THE ONLY PORTION OF THE GLAND IN WHICH THERE WAS ANY OF THE NORMAL TISSUE REMAINING WAS IN THE THINNED AND GREATLY INFILTRATED CORTEX.

greatly compressed and pushed downward. It measured about 4 centimeters in length by 2½ in width and was very pale on section. The suprarenal gland consisted of a sac with walls about 2 mm. in thickness, and was entirely filled with blood, the greater portion of which was clotted. There was some liquid blood occupying about one-third of the sac on its upper and inner portion. The walls of the cavity containing this were ragged and covered with shreds of fibrin. The balance of the sac was filled with an exceedingly firm organized clot,

which had unquestionably existed prior to birth. The portion of the liver and diaphragm to which the upper portion of the mass was adherent were removed with it; just anterior to the margin of this adhesion there was a slit in the sac, which was evidently the point at which rupture had occurred, resulting in a fatal secondary hemorrhage into the peritoneal cavity. The hemorrhage had also penetrated the peritoneal covering of the diaphragm and infiltrated the muscular tissue to some extent. The rupture into the perirenal tissues had evidently antedated the peritoneal rupture, as was evidenced by the firmness of the clot. The left suprarenal gland was normal; the left kidney showed very beautiful uric acid infarcts but was otherwise normal. The spleen and liver were slightly congested but showed no hemorrhages. The ureters were normal and the bladder empty. The pancreas, stomach and intestines were normal and the hepatic duct was patulous. The thymus was normal. The lungs were slightly congested along the lower border. There were no hemorrhages into any of the thoracic organs and no abnormal degree of congestion. In examining the heart the right auricle was found to contain a few jelly clots. The left auricle and both ventricles were empty, the valves and muscle were normal. The brain and cord were not examined. There were no hemorrhages into the muscular tissue. Cultures made from the heart-blood, kidney, liver, spleen and umbilical cord remained sterile.

The microscopic examination was kindly made for me by Dr. C. Y. White, Assistant Director of the Pepper Laboratory of Clinical Medicine, and is as follows:

"*Suprarenal.*—Its capsule is thickened and in many places its fibers are separated by extravasated blood. In the interspaces there are many free brownish granules, and in some of the cells (chiefly leucocytes) there are areas showing round cell infiltration. The cortex is the only recognizable part of the gland; in many places directly under the capsule it shows round cell infiltration. The trabeculæ of the gland are swollen and seem to have undergone hyaline degeneration. The cells of the gland are indistinct, swollen and homogeneous. The nuclei are pale with very little structure visible. Rarely is there seen a cell showing the usual fatty appearance of the normal cell as it appears in the adult. Between the few remaining cells and separating them from each other there is marked evidence of hemorrhage in the form of irregular masses of granules, some of

which are free, some in the cells. It is not improbable that they are in part due to the formalin fixation. Only in one section and in a very limited area did this pigment give the iron reaction. There were a few hematoidin crystals here and there throughout the gland. The portion of the diaphragm to which the gland was attached showed the same granules described above between the bundles of muscle fibers and in places throughout the fibers. The adhesions connecting the gland with the muscle showed marked extravasation of blood and the presence of granules.

"*The liver* shows slight fatty infiltration with some evidence of fatty degeneration of the cells, and probably a little more connective tissue than is normal.

" *The kidney* shows cloudy swelling, in some places going on to desquamation of the lining cells of the tubules. The cellular tissues between the suprarenal and the kidney show marked extravasation of blood."

CASE II.—An infant born at term after a long, natural labor, was apparantly normal at birth. On the third day its temperature became elevated and continued up, reaching 104.2° F. on the day of death, the ninth day. At the autopsy there was noted a papular eruption over the neck, shoulders and arms. Both suprarenals showed slight hemorrhage into the cortex. There was present some congestion of the pyramids of the kidneys; congestion of the spleen and of the bases of both lungs; some enlargement and congestion of the bronchial glands; an enlarged liver, yellowish in appearance, microscopically showing fatty infiltration and degeneration; extensive hematoma over the right posterior parietal, and right upper occipital regions, and a subperiosteal hemorrhage, the size of a half-dollar, in the mid-occipital region. Cultures made from the liver, kidneys, spleen, suprarenals and heart-blood remained sterile.

CASE III.—An infant dying on the fifteenth day showed at the autopsy, scattered over almost the entire body, small reddish papules, which in some areas were pustular. On the flexor surfaces of the arms there was a purpuric eruption. Over the lower lumbar region, slightly to the left of the vertebral column, there was a healthy-looking wound, which had been caused by the removal of a congenital tumor. The infant had double club-foot; its liver was enlarged and yellowish; the suprarenals a little enlarged, showing on section a central sac containing a small amount of brownish, grumous material having the appearance of altered blood. The walls of the sac were

smooth; there was no hemorrhage into the cortex. The right lung was slightly congested at the base. Cultures made from the heart-blood showed a pure growth of a short, somewhat oval, rather thick bacillus, which responded to the various tests for the colon bacillus. A microscopic examination of the gland was unfortunately not made in this case, but the appearance of the contents of the central sac was so suggestive that it was considered a case of hemorrhage.

A very extended and careful search of the literature has enabled me to discover references to 87 additional cases. Of this number 19 are merely referred to, 8 by Mattei, 3 infants and 5 still-born, and 11 by Still. In many others the reports are meager and in the vast majority they are incomplete. The fact that as many as 44 of these 87 cases came under the observation of three men, Spencer 23 in 105 autopsies, Mattei, 15 in 61 autopsies and Riesman 6 in a smaller number, would seem to indicate that the position taken by Spencer and Mattei that congestion of some degree is almost always present, and that *hemorrhage into this organ is common,* is probably correct. The three cases communicated by myself were observed in 9 autopsies made during the past winter on infants dying within the first few days of life, a fact which is further evidence in support of this view. Instances of extensive hemorrhage existing as practically the only lesion, destroying nearly all the tissues of the capsule forming a large blood-cyst with rupture into the peritoneal cavity, and causing death from loss of blood, conditions which existed in the first of my cases, are not common. Tuley, Milroy, Prudden, Hodenpyl, Droubaix and Hervey have reported cases of this description. Several others (among them Mattei, Spencer and Parrot) have reported cases in which there was rupture with slight escape of blood into the peritoneum and perirenal tissues, but in each of these there was extensive hemorrhage and congestion of other organs, the suprarenal lesion being but a small part of the general process.

ETIOLOGY.

Many opinions have been expressed regarding the cause of hemorrhage into the suprarenal gland. Milroy[8] has suggested that owing to the congenital weakness of the veins any undue venous obstruction results in rupture of the vessels and escape of blood into the tissues of the gland. A practically similar view

has been expressed by Bissell.[4] Still[5] considers the condition traumatic in some still-born children and in those dying a few days after birth. In a yet larger proportion he believes it due to asphyxia (delayed respiration at birth producing intense venous congestion and consequent hemorrhage into the naturally lax and highly vascular tissue of the suprarenal gland). After the fifth day it is probably due to some change in the walls of the vessels, such as acute fatty degeneration. In later cases he considers a toxic cause possible. Lewes[6] agrees with Still in believing that the morbid condition of the vessel walls is responsible for the hemorrhage in some instances. Mattei[1] believed that the firm contraction of the uterine muscles and the resistance of the parts traversed led to compression of the inferior vena cava between the relatively large liver and the vertebral column, thus damming back the blood into the soft non-resistant tissues of the suprarenal gland. In some instances this resulted in congestion only, in others the vessels ruptured and hemorrhage occurred. Under normal conditions these causes acted sufficiently to induce congestion, and when the uterine contractions were excessive or the resistance of the parts was unduly great, hemorrhage resulted. Spencer,[2] whose experience in making autopsies on still-born children has been very extensive, considers the normal delicacy of the walls of the fetal blood-vessels the one essential factor of hemorrhage into the suprarenal gland or into any of the viscera. He thinks it possible that syphilis, by further weakening the vessels, may increase the liability to hemorrhage. Asphyxia livida, while not a potent factor in itself, will in the presence of other causes increase the severity of the hemorrhage, and it is not impossible that a vasomotor influence may be exerted upon the vessels, as the result of injury to the central nervous system, which may contribute something to the production of hemorrhage. He believed, however, that mechanical squeezing of blood into the part during the process of labor and external violence, such as pressure of the hand in making traction, rupturing the vessels at the part pressed upon, are much more commonly responsible than any form of asphyxia. In most of his cases some sort of interference was necessary to complete the labor, the majority being deliv ered by the lower pole. From this he concludes that delivery by the lower pole, especially when traction is used, greatly predisposes to hemorrhage into the suprarenal gland. He found

no evidence to show that infection was responsible for the condition in still-born children. He does, however, believe that such a cause is sometimes active in young children. Rayer[7] agrees with Spencer in considering fragility of the veins essential to the production of suprarenal hemorrhage, but in addition he lays stress upon the want of firmness of the medullary substance. Parrot[8] attributes congestion and hemorrhage of these organs to their abundant blood supply, their intimate relation to the inferior vena cava, and as a final factor, the circulatory troubles so common during fetal life and during labor. Fiedler[9] considered traumatism and disturbed nutrition the principal causes. In his two cases, included in the subjoined series, there was marked fatty degeneration of the suprarenals, especially of the medullary portion, to which condition he attributed the hemorrhage. Merkel[10] thinks that syphilis acts as a causative factor, and in commenting on the two cases reported by Fiedler suggests that possibly they were of syphilitic origin. Duckworth[11] considers acute inflammation of the tissues and convulsive seizures sometimes active. Hutinel[12] believes that congestion and hemorrhage are produced in the following manner: The glands are made up of a very rich venous network. These veins terminate in a large principal trunk, emptying on the left side into the renal vein and on the right directly into the vena cava. In case of venous engorgement or thrombosis of either the renal vein or the inferior vena cava, the blood is dammed back into the capsule causing either congestion or hemorrhage. Droubaix[13] confirms this view and also supports the views expressed by Mattei. Demelin,[14] in discussing the etiology of hemorrhage in the new-born, of which hemorrhage into the suprarenal capsule is sometimes a part, suggests the following causes: (1) Traumatism; (2) circulatory troubles in connection with the establishment of the pulmonary function; (3) infections and dyscrasias. The traumatic factors are usually the obstetrical operations, especially delivery by the breech, traction, and the frictions and flaggellations used to resuscitate the apparently dead-born. The circulatory troubles are usually favored by congenital weakness of the vessels which makes it difficult for them to resist the sudden tension they experience at the moment the infant begins to breathe. Townsend,[15] as causes of hemorrhage in the new-born, recites those given by Demelin, and mentions further hemophilia, plethora, debility, retention of

meconium and too early ligation of the cord, a cause also mentioned by von Kirvisch,[16]; but he considers as much more common than all these some form of infection, such as syphilis, the acute infectious diseases or septicemia from infection of the cord. He quotes Klebs and Eppinger[17] as having isolated a microorganism in some cases of hemorrhage in the new-born which they called "mona hemorrhagica," and he relates von Ritter's experience in the Prague Foundling Asylum Hospital in which he succeeded in practically exterminating the condition which had formerly been prevalent by the establishment of newer and larger wards. Gaertner[18] has contributed further evidence in support of an infectious origin by isolating a bacillus which somewhat resembled the colon bacillus, but which could be readily differentiated from it. He was able to cause visceral hemorrhages in dogs by introducing this organism into the peritoneal cavity and to recover it from the blood of the animals. There has also been abundant experimental evidence adduced in support of the infectious origin of hemorrhage in the newborn, and of hemorrhage into the suprarenal gland in particular: Thus, Roux and Yersin[19] by injecting diphtheria bacilli under the skin of rabbits, guinea-pigs, and pigeons induced general dilatation of the vessels, congestion of the intestines and kidneys, and almost constantly in guinea-pigs, congestion of the suprarenal glands. Langlois and Charrin[20] recognizing the important role which the suprarenal glands play in the chemistry of the body concluded that they were probably seriously affected by the various infections. In order to confirm this opinion they made a careful study of a guinea-pig dying from an acute pyocyanic infection. They noted that the suprarenals were increased in size, that they were deeply congested and their normal pigment increased. Microscopically the central zone was engorged with blood, the vessels dilated and in some areas there was hemorrhage. The cells contained numerous colored granules which did not exist normally. Cultures made from the organ showed the bacillus pyocyaneus. They also demonstrated that the condition was a true intoxication in inducing it by injecting the soluble bacterial products, as well as by injecting the bacillus. Pilliet[21] confirmed the toxic nature of the lesion by securing exactly similar results from the subcutaneous injection of the essence of cloves into rabbits and guinea-pigs. Roger,[22] by inoculating guinea-pigs with a virulent culture of

Friedlander's bacillus found that at the end of from twenty-four to thirty-six hours the suprarenal glands were enlarged, ecchymosed, and sometimes the organ was entirely filled with a bloody effusion. On section the parenchyma seemed transformed into a bloody mass, only a few small areas in the periphery of the organ remaining intact. He agrees with Langlois and Charrin in making the soluble bacterial toxins responsible for the bleeding. Orlowski[25] considers the various varieties of hemorrhages in the new-born to be due chiefly to syphilis and other infectious conditions, the most common agents being the streptococcus, the staphylococcus and the colon bacillus, the main point of entry of the organism being a poorly tied umbilical cord.

In summing up the various views expressed we find the following list of causes: (1) Weakness of the vessel walls, normal or abnormal; (2) traumatism, especially during labor from pressure of the hands in making traction in delivery by the lower pole, and from the frictions and flaggellations used to resuscitate the apparently dead-born; (3) asphyxia from delay in the establishment of respiration at birth; (4) acute fatty degeneration of the vessel walls; (5) fatty degeneration of the tissues of the gland; (6) firm contraction of the uterine muscles, the resistance of the parts traversed, and consequent compression of the inferior vena cava between the liver and the vertebral column, thereby producing congestion and hemorrhage into the non-resistant tissues of the suprarenal gland; (7) convulsions; (8) syphilis; (9) central vasomotor influence from cerebral lesions; (10) mechanical squeezing of blood into the part during the process of labor; (11) too early ligation of the cord; (12) arrest of the circulation through the umbilical artery from the compression of the cord or separation of the placenta; (13) thrombosis of the renal vein or inferior vena cava; (14) infection.

The consensus of opinion seems to be against syphilis as a very active factor. It was not seen in any of the cases here reported. It is probable that this condition, together with the influence of hemorrhage and other brain lesions acting upon the vasomotor center, if they act at all, act only as predisposing factors. Thromboses of the renal vein or inferior vena cava seem to have been of infectious origin in the cases in which they were noted, so that the last two causes mentioned may practically be classed as one. We have left, therefore, eleven causes,

any one of which may at sometime be in part or entirely responsible for the production of hemorrhage into the suprarenal gland.

The position of Spencer, and those who support him, that the fundamental element in the production of suprarenal hemorrhage is the weakness, normal or abnormal, of the vessel walls, must be accepted without question. Traumatism is doubtless the sole cause of the condition in some instances, and a contributive factor in a large majority of the cases. Where other causes may have acted to induce congestion the occurrence of injury to the gland or parts adjacent to it is liable to produce hemorrhage, and in the series of cases attached there are many instances of this course of events. Asphyxia has been very infrequently noted in the cases here reported, and as Spencer has suggested it is probably a factor of no great moment. It should be borne in mind, however, that asphyxia produces increase in the blood pressure, and anything which acts to elevate the pressure in the delicate vessels of the new-born will necessarily increase the tendency to bleeding. Microscopic examinations have shown no instances of fatty degeneration of the vessel walls, but in two cases reported by Fiedler (XVI and XVII) there was marked fatty degeneration of all the other tissues of the gland. The view expressed by Mattei, that firm contraction of the uterine muscles and the resistance of the parts compress the inferior vena cava between the liver and the vertebral column, thereby producing congestion and hemorrhage into the non-resistant tissues of the suprarenal gland seems to be reasonable. In case XV these conditions were present associated with a cord wrapped once about the neck.

These factors probably contribute something to the occurrence of hemorrhage in the majority of cases. Convulsions were noted in five of the cases (XII, XIV, LIX, LXX and LXXVII). It is impossible to indicate the amount they contribute, but it is easy to understand how in the presence of severe congestion a violent convulsive seizure might give rise to hemorrhage into the suprarenal gland as well as into other organs. It has been demonstrated by Spencer that mechanical squeezing of blood into the abdominal viscera and especially the suprarenal gland, during labor is liable to occur in delivery by the lower pole. Too early ligation of the cord has not been noted in the collected cases, but compression of the cord as the

result of prolapse has been recorded once (Case XII) and as the result of the cord passing around the neck, three times, (XV, XXXVIII and LIII). An infectious origin for the condition has received fairly definite support by positive bacteriological findings in four cases observed by Riesman (XXVI, XXVII, XXVIII and XXXI), in which the staphylococcus aureus and albus were grown in pure culture from the blood and tissues. In some other instances (Cases I, II, III, XIX, XXIX, XXX, LXI and LXVII) the evidence is strongly in favor of an infectious origin, and it is not improbable that some form of infection is at the bottom of the trouble in still other cases. In Case No. I, for instance, notwithstanding the negative bacteriological examination, the presence of a mild papular skin eruption during life and the discovery of marked cloudy swelling of the kidney and fatty infiltration of the liver with some beginning fatty degeneration of the cells have led to the conclusion that this case was probably due to some form of infection. It should be remembered, however, that these changes might be dependent upon some toxin bearing no etiological relationship to the suprarenal hemorrhage, which fact makes a definite statement as to the etiology impossible. The results here obtained, together with the positive findings of Klebs and Eppinger, and Gaertner in cases of hemorrhage in the new-born, and the abundant experimental evidence of the power of bacteria to produce hemorrhage into the suprarenal capsule would make the existence of such a cause as this unquestionable, and I am inclined to believe that if complete histological and bacteriological studies had been made in other cases that striking evidence of a toxic origin would have been found. If such a course be applied to all future investigations it will doubtless be shown that this is not only a possible but by far the most common cause.

The act of vomiting seems to have been responsible for hemorrhage into the right suprarenal gland in an apparently healthy twelve and a half hour old infant under the care of Milroy (Case V); there was no other lesion found. It is probable that congestion of the gland had existed previously. Severe paroxysms of pertussis were held responsible for the hemorrhage in a two months old infant observed by Duckworth (Case LXXII), and Still found a hemorrhage into the left suprarenal in a case of miliary tuberculosis in a fourteen months old infant (Case LXXIII, in which there were tubercles present in the

suprarenal gland). Churton (Case LXXV) reports a case of hemorrhage into both suprarenals in a child dying as the result of surface burns. The hemorrhage in this later case may have been due to the action of some retained toxic product, to vasomotor disturbance from reflex excitation or to embolism.

To summarize briefly: The most common causes in still-born children are probably prolonged and difficult labors, those requiring manipulation, and especially those requiring delivery by the breech. In some infants dying within a few days of birth the lesion may still be attributable to injuries inflicted during labor, but in a vast majority of these, some form of infection is responsible, while in practically all cases dying after the tenth day some form of infection produces the condition. The other causes mentioned act alone occasionally and frequently contribute something toward the fatal termination.

Classification.—Still makes the following classification of these cases: (1) Those in which death occurs within a few hours or days of birth, never later than the sixth day; (2) those in which death occurs later and the suprarenal lesion is a complication of some disease, usually of the respiratory tract; (3) those in which after an acute illness of two or three days, usually with a purpuric or bullous eruption, death occurs, and the suprarenal lesion seems to be a part of the fatal disease. The following classification having some relationship to the probable etiology of the condition at different periods of its occurrence would seem more appropriate: (1) Those in which death occurs before or during labor (still-born), due chiefly to traumatism from manipulation; (2) those in which it occurs between birth and the detachment of the stump, due chiefly to infection through the cord, and (3) those dying after detachment of the stump, usually of an infectious or toxic origin.

Dividing the 90 cases forming the basis of this paper according to this classification, it is noted that 28 were observed in still-born children, 27 between the date of birth and the sloughing of the cord and 11 during the third period. Of the remaining 24 cases in which the date of birth is not definitely indicated, it seems probable that 11 would fall under the second classification and 2 under the third. In the others it is impossible to reach any conclusion. This finding establishes the fact that the vast majority of cases occur either before or during labor or within the first few days of life.

PATHOLOGICAL ANATOMY.

The hemorrhage may be unilateral or bilateral. In the 65 cases in which its location is mentioned it was unilateral 25 times, on the right side 15, on the left 9, not stated once; and bilateral 43 times, being greater on the right side 5 times and greater on the left twice. In the 36 remaining instances the side of greater prominence is not indicated. These findings would seem to confirm the view long since expressed that the lesion is more common on the right side than on the left. The explanation made by Mattei to account for this is that this gland is more liable to congestion or hemorrhage on account of the direct emptying of its capacious veins into the inferior vena cava, and its greater liability to pressure in consequence of its anatomical position, lying as it does between the liver in front and the vertebral column behind.

To the naked eye the glands present a varied appearance. Where the hemorrhage is large there may be a tumor the size of a duck's egg occupying the region of the suprarenal gland, having a reddish-brown or black color with a smooth, glistening surface, its general appearance suggesting an enlarged hemorrhagic kidney. After rupture into the peritoneum, as is apt to occur where the gland is much distended, there will be found, usually on the upper anterior surface of the mass just below its point of contact with the diaphragm, a small, slit-like opening. The overlying colon and duodenum on the right side, or the descending colon, spleen, pancreas or stomach on the left may be adherent to the sac. The size of the gland depends on the extent of the hemorrhage, and the external appearance on the extent and location. If the organ is well distended, the cortical substance thin and yet intact, the normal yellowish tinge will be somewhat reddened. Where the hemorrhage is small and central, aside from moderate enlargement the gland may appear normal. Occasionally, as in case LVII, it may be hemorrhagic in only one portion of the organ, giving it an irregular, more or less lobulated or club-like appearance. In one instance there was an isolated hemorrhage about the size of a nickel immediately beneath the fibrous capsule. Occasionally small ecchymotic areas are scattered over the surface of the gland. On section it is seen that in the vast majority of cases the hemorrhage is mainly into the medullary portion. Usually its tissues are

entirely infiltrated and frequently completely destroyed, leaving a large cavity, the walls of which are formed by a flattened and infiltrated cortex, and sometimes, as in my own case, there may be scarcely any of the cortical tissues recognizable. The sac is usually filled with dark liquid or clotted blood; the clot may be organized in parts, grayish in color, and in striking contrast with the deep red of its more recent portions, producing a more or less mottled appearance. After the blood escapes the walls of the cavity show a ragged appearance, due to the adherence of particles of clot and shreds of fibrin. Where the hemorrhage is small and recent the walls are smoother. As in some cases the hemorrhage appears in the medulla in the form of scattered areas (Cases LIV and LXV), so also one occasionally finds a number of small blood cavities distributed here and there throughout the gland (Case XXXVII). Section has in several instances given the appearance of a small cyst containing grumous-looking material, having the appearance of altered blood, and in such cases, without a microscopic examination, the condition is liable to be considered a *post-mortem* change. Hemorrhage is rarely limited to the cortex, and when it is it occurs in the form of scattered ecchymotic areas, usually visible on the surface of the gland. Rupture into the peritoneum, the perirenal or post-peritoneal tissues is quite common, having occurred in 11 of the cases. Occasionally the peritoneal hemorrhage is very abundant. In one instance rupture occurred into the substance of the liver, the hemorrhage slitting up Glisson's capsule for a considerable distance, and in Case No. I the hemorrhage penetrated into the tissues of the diaphragm. Hemorrhage into the cellular tissues surrounding the gland and kidney sometimes occurs irrespective of rupture. Thrombosis of the renal vein and the inferior vena cava has been noted in several instances. Pathological changes other than hemorrhages have been recorded in 2 cases reported by Fiedler, and in a case reported by Still, a case of miliary tuberculosis in a fourteen-months old infant, there were one or two gray tubercles in the substance of the gland.

PATHOLOGICAL HISTOLOGY.

Microscopical studies have been made in very few of the cases, but four times in infants dying within the first few days of life, and three times in infants of fifteen months or older. In 2 cases reported by Fiedler (XVI and XVII) there was found,

in addition to the evidences of hemorrhage, extreme fatty degeneration of all the tissues, the vessel walls alone remaining free. It is recorded that no fat-free cells were found. In Droubaix's case (XII) it was merely noted that the vessels were obstructed by blood from the periphery toward the center. The cortical substance was nearly preserved in all of the sections, there being only a few isolated areas of infiltration as the medulla was approached. In Still's case (LXXIII) the medulla was infiltrated with extravasated blood corpuscles but there was no disintegration of the tissues. The hemorrhage was nowhere circumscribed. The cortex showed enlargement of the small blood-vessels and some extravasation of blood, but less than in the medulla. Andrew's case (LXXIV) showed a uniform diffuse extravasation of blood, the normal tissue elements being diminished in number. In Garrod and Drysdale's case (LXXIX) the stroma was fairly well preserved, the cells enclosed in its meshes had largely disappeared, being replaced by effused blood, some of the individual spaces being entirely filled by red blood corpuscles. In some areas the cells were present in considerable number, the nuclei staining well, but the cell substance being practically destroyed. The microscopic examination made in my first case has been given above in detail, and probably expresses the microscopic findings as definitely as any description that could be given. As a matter of fact, the results of the microscopic examination, in the event of uncomplicated hemorrhage, will vary according to the degree, location and age of the hemorrhage. Aside from this difference it will be practically the same in all cases. The special advantage of a histological study lies in the possible discovery of some pathological condition, either in the suprarenal or some of the other organs, which may throw light upon the etiology. In my own case, as pointed out above, the histological findings entirely altered the view which had been taken regarding the cause of the hemorrhage.

SYMPTOMATOLOGY.

There have been no symptoms in the cases reviewed which seem in any way related to the lesion. In Case No. I, the child was normal until the evening of the second day. It was then seized with sudden pallor, elevation of temperature, restlessness, crying as if in pain, and shortly before death with

cyanosis and rapid, labored respiration. Symptoms of collapse were noted in several of the cases in which rupture had occurred. In Case VIII a dirty color of the skin had been noted before death.

DIAGNOSIS.

In cases in which the hemorrhage is small, and especially if limited to a single capsule, it is probable that symptoms and signs would be absent. Rayer has suggested that the presence of a gland large enough to be palpated, in the absence of symptoms suggesting lesions of other organs, might lead to the suspicion of a suprarenal hemorrhage. Sudden symptoms of collapse without evidence of hemorrhage into other organs, especially if a tumor has been located in the suprarenal region, might suggest the possibility of this condition. In the opinion of the majority of observers, however, the lesion is considered impossible of recognition in the cases occurring shortly after birth.

The case of Wainwright (LXI) would suggest that the lesser degrees of hemorrhage sometimes go on to organization without softening. His case occurred in an infant dying at the age of two months of bronchopneumonia. The autopsy showed the remains of an old hemorrhage into the medulla of one suprarenal. The cortex was normal to the naked eye and nearly so microscopically. Similar cases probably occur with greater frequency than statistics indicate, and it is highly probable that they lead to changes in the gland which seriously interfere with its important function. Neusser[84] has pointed out that hemorrhage may lead to cystic or fibrous change in the suprarenal gland, and as marked fibrosis of the gland is sometimes the nature of the lesion in Addison's disease it is not unreasonable to suppose that it may sometimes have had its origin in hemorrhage at an earlier period. Through reduction of the vitality of the gland it would be rendered more susceptible to infection by the bacillus tuberculosis and it is well-known that in a fairly large proportion of cases of Addison's disease this lesion is associated. The relationship between hemorrhage and the various new growths which affect these glands is rendered uncertain by our lack of knowledge of their etiology, but there has been no evidence adduced to confirm the suspicion that such an association might exist.

The immediate effect upon the economy is dependent upon the degree of hemorrhage. In the milder non-infectious forms, as has been intimated, it is probably nil. These organs play a very important role in the chemistry of the body. Evidence of this exists in the fact that rapid death follows either their removal or the ligation of their vessels. The exact nature of their function is not definitely determined, but it is probably in part excretory, and certainly in part secretory. In any event the probable effect of a large destructive bilateral hemorrhage would be to produce a cessation of its function and lead to a rapidly fatal autointoxication. Where hemorrhage into the gland is extreme and it is much distended, rupture into the post-peritoneal or perirenal tissues or into the peritoneum may occur and give rise to sudden death. This termination has been noted several times in the attached cases. Mattei quotes Lobstein as saying that where the volume of the hemorrhage into the suprarenal gland is large sufficient pressure may be made upon the semi-lunar ganglia and solar plexus (situated between the crura of the diaphragm and supported by the vertebral column) to cause death. Since the solar plexus is the central point from which the nerves supplying the abdominal viscera converge and diverge, it is evident that any shock or irritation of this plexus may result in the paralysis of organs whose functions are essential to life. Brown-Sequard observed arrest of the heart's action in consequence of the bruising of one or the other semi-lunar ganglia, especially the right, and Mattei has noted the same result in the rabbit. It has been suggested by Stengel that in the light of our knowledge of the influence of the suprarenal gland upon blood pressure, in some cases of hemorrhage in which the lesion has not been sufficient to entirely destroy the circulation of the gland, there may escape into the general circulation a sufficient amount of suprarenal substance to elevate the blood pressure and thereby give rise to widely disseminated hemorrhages such as are induced by the increased blood pressure in cases of asphyxia. This may account for the widely distributed ecchymoses present in some of the recorded cases.

REPORTED CASES.

CASE IV.—Tuley (ARCHIVES OF PEDIATRICS, November, 1892). A male child of a healthy mother. Labor was very short and unassisted. There was no history of injury. The temperature

at birth was 99.6° F. The child failed rapidly. It had mild jaundice on the third day; this became intense before death. The temperature became elevated to 104.4° on the third day; on the evening of the fourth day the expression was anxious and painful and the face was drawn. The respirations increased to 72 and were sighing and labored. The pulse was weak and beat 200 to the minute. The extremities were cold. The abdomen was tense. The child died on the evening of the fourth day.

Autopsy.—The brain was not examined. The lungs were imperfectly crepitant, rather firm and heavy. On opening the abdomen about one ounce of fluid blood escaped. Several large clots were discovered in the left iliac region; 8½ ounces of fluid and clotted blood were measured. The liver was displaced upward by the right kidney. The left kidney was a little pale; the right was enveloped in a large mass of clots filling the right side of the abdomen. The right suprarenal gland was almost as large as a goose egg, and was distended with fluid blood and clots. The kidney was much compressed but normal in appearance. The hemorrhage into the suprarenal gland had ruptured secondarily into the cellular tissue surrounding the kidney, into the post-peritoneal tissue and into the peritoneal cavity. The rupture occurred at the apex of the hemorrhagic sac.

CASE V.—W. F. Milroy (*Amer. Jour. of Obstet.*, July, 1884, Vol. XVII, p. 772). A male child; mother healthy; no history or evidences of syphilis. The labor was normal and short. The child was in excellent condition at birth and for twelve and a half hours seemed perfectly normal. It was then seized with vomiting, which lasted for a few minutes. Rapidly following this the extremities became cold, the features pale and sunken, the respirations shallow and rapid and expiration accompanied by a sharp cry. The child died in one hour and thirty minutes after vomiting. There was no history of violence subsequent to delivery.

Autopsy.—The only pathological condition present was as follows: The intestines were displaced to the left by a firm blood-clot about the size of a man's fist which entirely enveloped the right kidney and right suprarenal gland. A large quantity of fluid blood was found within the peritoneal cavity which had escaped from an opening into a hemorrhagic sac which proved to be a hemorrhagic right suprarenal gland.

CASE VI.—Milroy (*Ibid*) refers to a case spoken of to him by Dr. A. E. Maxwell. In an autopsy on an infant Maxwell had found a bilateral hematoma of the suprarenal glands.

CASE VII.—Prudden (*Proceedings of New York Path. Soc.*, 1899, p. 92). The author showed specimens from an infant born after a normal labor. The child breathed badly and performed its general functions badly from birth to the time of death on the fifth day.

Autopsy.—The abdominal cavity was filled with dark fluid blood or bloody serum, and a large and old clot lying over the region of the right suprarenal capsule. Both kidneys and the remaining abdominal viscera appeared normal, save that closely surrounding the right suprarenal gland there was a dense, firm, ovoidal blood clot about 4 centimeters long, 3 centimeters wide and 2.5 centimeters thick. The suprarenal gland was apparently unchanged save by pressure, and a considerable infiltration of blood in several places.

CASE VIII.—In the discussion of the former case Dr. Northrup referred to having seen a child in whom one suprarenal capsule showed hemorrhages with quite extensive destruction of the gland tissue. Hematoidin crystals were found in his case, and a dirty color of the skin had been noted before death.

CASE IX.—Hodenpyl (*Proceedings of New York Path. Soc.*, 1890, p. 67). The infant, born after a perfectly normal labor, did well until within a few hours of death, which occurred on the third day. The symptoms were those of sudden collapse.

Autopsy.—The abdominal cavity was filled with blood which came from a ruptured hemorrhagic right suprarenal gland. The remarkable feature of this case was that the suprarenal hemorrhage had penetrated the under surface of the liver and had stripped up Glisson's capsule for a considerable distance. There was also an infiltration of the lung. No other lesions were found.

CASE X.—Gueniot (*Bull. de la Soc. anat. de Paris*, Vol. XL, p. 182, 1865). The author presented the suprarenal capsules from a new-born infant. Their cavities were filled with blood. During the labor there occurred prolapse of the cord. The forceps were used to shorten labor. The infant was apparently dead when born. Fetal movements had been felt less than two

hours before birth. There had not been any direct pressure which could be made to account for the hemorrhage. No other lesions were noted.

CASE XI.—Hervey (*Bull. de la Soc. anat. de Paris*, Vol. XLV, p. 263, 1870), showed specimens from an infant dying suddenly on the tenth day. Up to the morning of the same day it had been well. There was very little evidence of trouble up to within a few minutes of death.

Autopsy.—A serosanguinolent fluid filled the entire abdominal cavity. There were some fairly dark fibrinous clots present. The kidneys showed no naked eye changes. There was a bloody fluid in the ureters, and there were many ecchymotic areas in the adipose capsule of the kidney. The suprarenal capsule on the left side was reddish in its medullary portion and on section was very congested. The right suprarenal was a huge black mass; it was converted into a cavity more or less regularly limited by the cortical substance and contained blood-clots. There was a slit in the peritoneal surface about 1½ centimeters long, immediately under the liver, which was occupied by a small clot. This was evidently the point of origin of the hemorrhage into the peritoneal cavity. There was a bloody infiltration into the osseous tissue of the occipital region. All other organs were normal. There was a history of the infant having fallen from bed two days before death. The fall was thought to have been on the head and back; it was the probable cause of the hemorrhages.

CASE XII.—Droubaix (*Thèse de Paris*, 1887). The infant was delivered by podalic version in an apparent state of death; it was resuscitated, however. There had been a prolapse of the cord. The whole labor had occupied forty-five minutes and the version was done rapidly. The infant was well nourished. There was a slight marginal placenta previa. The cord was 45 cm. long. The infant had a convulsion eleven hours after birth. It died on the third day.

Autopsy.—Aside from the suprarenal capsules the organs contained nothing abnormal. The capsule on the right side was almost completely destroyed and replaced by a hemorrhage, making a mass the size of a hen's egg. The blood had escaped into the surrounding cellular tissues. The hemorrhagic capsule was composed of a thin wall 1 mm. in thickness. The renal vein was free and presented no clots. The left capsule was

considerably increased in size. It was very congested, almost black in color in the portions above and in front of the kidney. The capsule was not broken. There was, however, a focus of blood in the cellular tissue posterior to the capsule, and over the upper portion of the kidney. There was a small, fibrinous clot in the renal artery floating free in the vessel. The renal vein was completely obstructed by a clot adherent to the walls which on section was partly fibrinous and partly jelly-like. On section of the capsule the medullary portion was found completely disorganized and replaced by a focus of blood. On microscopic examination, from the periphery towards the center the vessels were found to be obstructed by blood. The cortical substance was nearly preserved in all the sections; there were a few isolated infiltrations which increased as the center of the mass was approached.

CASE XIII.—Valleix (*Clinique de maladies des enfants nouveaunes*, Paris, 1838). The infant died on the seventh day after birth. The autopsy was held two hours after death. The kidneys were of normal size; their veins were engorged with blood. The suprarenal glands were much enlarged. The cavity of the left was the size of a small hen's egg, and contained a reddish liquid and a large quantity of fibrin. The contents had the appearance of boiled bloody liquid. This material seemed to be deposited on the walls of the cavity and formed a covering a half-line in thickness. It was strongly adherent and seemed organized. The walls of the capsules were reddish and easily torn. The right capsule preserved its yellowish color and was less distended. It contained a small black blood clot half an inch in thickness. The walls of the capsule were thick. The clot was easily shelled out.

CASE XIV.—J. Parrot (*Archives Générales de Medicine*, 1872). A female infant, on the second day of life developed convulsions. This was followed by a comatose condition; the rectal temperature was 33.4° C. During the third night the symptoms increased and death occurred on the following morning.

The autopsy showed a child fully developed. The lungs were congested in the dependent portions. There was some cerebral congestion and the brain was soft. There were soft clots in the cavities of the left heart, and some firm clots in those of the right heart and in the pulmonary artery. There

were some rather grayish looking masses in the right ventricle which were friable and not adherent to the walls which resembled the fragments of an old clot. They were composed almost entirely of leucocytes. There were small hematomata on the auriculoventricular valves and in the tunica adventitia of the aorta. Near to its origin was situated a small recent hematoma the size of a hemp seed. The right suprarenal capsule was distended by an enormous blackish-brown and friable blood clot; the left contained a much less abundant bloody effusion.

CASE XV.—Ahlfeld, (*Archiv. der Heilkunde*, 1870, No. XI, p. 491). Infant was born of strong, healthy primipara. Owing to the resistance of the lower uterine segment and the severe pains five warm douches and a subcutaneous injection of morphia were given. As the head passed through the vulva meconium passed with the liquor amnii. The heart sounds were not audible. The head was delivered by Ritgen's method. The cord was very long and wrapped once about the neck. The infant made one deep inspiration and then ceased to breathe. The limbs hung down relaxed. The child was finally revived. Twenty-four hours after birth it had an attack of suffocation. Three hours later a second attack occurred, and nine hours later a third and fatal attack.

Autopsy made sixteen hours after death. Female child, well nourished. The thymus was large and pale. On opening the abdomen there were seen two tumors the size of a hen's egg and having the appearance of huge extravasations of blood. They were found to be hemorrhagic suprarenal glands; on section fluid blood escaped. The kidneys were very large and congested, showing a deep bluish color on section. The origin of the hemorrhage could not be determined.

CASE XVI.—Fiedler (*Archiv. der Heilkunde*, 1870, No. XI, p. 301). A child born at term after a normal labor. The head was in the first position. The infant seemed normal for three days. It died after a few moments' illness on the fourth day, suffering from distension of the abdomen and severe dyspnea. There was no history of traumatism.

Autopsy.—There were no external evidences of injury. The abdomen was distended, the muscles well developed but pale. The subcutaneous tissues were rich in fat. The cranial cavity was not opened. The thymus was large and pale. The mucous membranes of the pharynx and esophagus were slightly

injected. There were extensive diffuse hemorrhages under the right costal pleura. There was slight circumscribed hemorrhage into the parietal pleura at the border of the right lower lobe. There were two small hemorrhagic spots on the anterior surface of the mitral valves under the endocardium. Four or five ounces of dark liquid blood were found free in the abdominal cavity. There was widespread congestion of the parietal peritoneum in the right half of the abdomen. The liver was pale. Both kidneys were pale, the right being displaced downward. The right suprarenal formed a circumscribed mass, the size of a hen's egg, and was adherent to the loose cellular tissue about the kidney. The ascending colon passed over the tumor, and was slightly adherent to it. On section its cortical substance was yellowish-red in color and moderately thick. On pressure a brownish-red substance escaped. The microscopic and mac roscopic study showed the tumor to be composed of blood clots which were not "fresh." The tumor on cross sections showed many pea-sized cavities which contained serum. The hemorrhage evidently sprang from the cortical substance of the gland. In many areas on microscopic examination there was free fat. The round and angular cells which were present contained fat cells and drops. No fat free cells were found. There was also fatty degeneration of the cells in the medullary and cortical substances of the left gland. The walls of the capillaries and larger vessels in both glands were normal. There was a thick dark red blood clot in the tissues surrounding the right kidney which entirely enveloped the latter and separated it from the greatly distended suprarenal gland. There was a hemorrhage into the medullary portion of the kidney, and the kidney was enormously distended. The cortical layer had apparently long resisted the pressure of the blood ; later it had burst and the blood had escaped into the capsule of the kidney; as the pressure increased the peritoneum was penetrated and there occurred fatal hemorrhage into its cavity. The vessels of the peritoneum showed no abnormality.

CASE XVII.—(*Ibid*). He refers to a prematurely born child dying a few minutes after birth, in which the autopsy showed atelectasis of both lungs, small extravasations of blood under the endocardium and into the mitral valve, extensive hyperemia of the brain, hyperemia of the intestinal mucous membranes and of the liver, and a very considerable swelling of both

suprarenals. This swelling was due to numerous punctiform hemorrhages. The cortical and medullary substances in this case also showed fatty degeneration.

CASE XVIII.—J. B. Bissell (*Amer. Jour of Obstet.*, Sept. 1894, p. 987). A male child. Healthy mother, multipara. The delivery was by podalic version, otherwise normal. Considerable gentle force was used in delivering the head and shoulders. Both arms were paralyzed; otherwise the infant seemed normal for two days after birth. It then became jaundiced and vomited. It had green mucus stools. On the third day the jaundice was very marked. On the fourth day the vomiting had ceased and the diarrhea was better. The child was very weak. Nourishment was given with difficulty. Death occurred from exhaustion on the fifth day. The temperature was elevated throughout, the maximum being 103.9°. The respirations reached 80 per minute.

Autopsy.—The liver was irregularly congested. It contained hard and rather whitish spots and streaks. There was a dark fluctuating mass the size of a kidney in the region of the right suprarenal capsule, and a similar tumor in the region of the left. On section about one ounce of dark fluid blood escaped. A thin, yellowish zone, with a broader reddish-black internal layer next to the top of the kidney, forming the wall of the blood sac, marked the remains of the suprarenal capsule. The mucous membrane of the intestine was much swollen and congested throughout its entire length. The gall bladder was distended with mucus. There was a small extravasation of blood in the pectoralis minor muscle but no bruise of the surrounding tissues and no fracture of the underlying ribs. There was also a slight extravasation in the tissues of the scalp over the right occipitoparietal region. There was nothing in the symptomatology to have suggested the condition found *post-mortem.*

CASE XIX.—Parrot (*Ibid*). The author observed a small, twelve-days-old infant which was very weak and suffering from thrush. Three days later it developed strabismus, intermittent trismus and stiffness of the legs. Death followed shortly thereafter. The temperature just before death was 34.2° C.

Autopsy.—There were enormous hematomata on the mitral and tricuspid valves. The muscular tissue of the heart was

slightly fatty. The umbilical veins and arteries had a mottled appearance and seemed to be fatty in areas. Throughout the entire abdominal portion of the inferior vena cava there was a clot from which prolongations extended into the iliac. At the level of the liver the thrombus showed areas of a reddish-gray color, being softened and purulent in the center. The thrombus extended into the renal veins completely obstructing that on the left side. The left suprarenal capsule adhered to the neighboring parts, notably to the pyloric end of the stomach and the pancreas. There was a bloody effusion into the post-peritoneal cellular tissues and in the anterior surface of the diaphragm. The whole capsule was distended by hemorrhage. There was a break in the wall of the capsule with an escape of blood into the parts above indicated. Both kidneys were very large and their surfaces blackish in color. On section liquid blood and venous clots escaped. The right capsule was healthy.

CASE XX.—Moissenet (*Jour. l'Expérience*, 1837. *Memoire de Rayer*) observed a new-born infant having an umbilical hernia.

The autopsy showed the right suprarenal to be the size of a kidney. On section there exuded serosanguinolent fluid. The cavity was covered by a pretty strong net-work of firmly coagulated fibrin of a brick-red color. There was a somewhat less extensive hemorrhage into the left.

CASES XXI and XXII.—Rayer (*Ibid*) also reports a case in a new-born, without giving the clinical history, which showed a large hemorrhagic tumor of one capsule, and relates another instance in a new-born in which the glands were transformed into pouches forming tumors in the lumbar region, covered with fibrin and containing sanguinolent fluid.

CASES XXIII AND XXIV.—LeConte (*Thèse de Paris*, 1897) reports two cases in which there was hemorrhage into both suprarenals in new-born infants.

CASE XXV.—Lancereaux (*Dict. Encyclop.*, 1875, T. 3, pp. 155 to 167) reports a case of an infant dying cyanosed two days after birth. The autopsy revealed a large clot in the right suprarenal gland, increasing it to four times its normal volume. There was a slight bloody exudate in the left suprarenal.

Riesman (*Post Mortem Records*, University Hospital, Philadelphia) made autopsies on six new-born children showing

hemorrhage of varying degree into one or the other of the suprarenal capsules. Through his courtesy I am able to include these cases in the series I have collected.

CASE XXVI.—No clinical history. Small new-born female infant. A small thrombus toward the umbilical end of the umbilical vein. The heart cavities contained fluid and clotted blood. There was slight congestion of the right lung posteriorly. The left suprarenal gland was normal, the right was very soft, slightly reddish, and on section showed a cavity containing bloody fluid. The mesenteric glands were enlarged, the liver congested and there was some ecchymosis in the mucous membrane of the stomach. There was imperfect ossification of the parietal bone. Cultures were made from the spleen and heart-blood. The result was not attached to the record.

CASE XXVII.—White female baby aged nine days. No clinical history. The cord was detached; a scab was attached to the navel which on removal showed a bloody surface. The abdominal cavity contained considerable blood on the left side. On this same side, occupying the left hypochondriac and lumbar regions, there was a tumor the size of a duck's egg. It lay behind the descending colon to which it was closely adherent. There was a large hemorrhage into the mesocolon which increased its thickness to three-eighths of an inch. The tumor mass was bluish in color, and on section proved to be a very large kidney and suprarenal. There was extreme hemorrhagic infiltration of the medullary substance of the kidney, the pyramids standing out as bulging black masses. The cortical substance was intensely congested. The suprarenal gland was enlarged. In its center was a cavity about the size of a walnut which was filled with blood. The walls of the cavity were soft and disorganized. The remaining portions of the suprarenal substance were soft and hemorrhagic. The capsule of the pancreas was hemorrhagic; the lungs were congested. The veins over the occipital lobe of the brain and in the Sylvian fissure were unduly distended with clotted blood. Cultures were made from the heart-blood and spleen; the result not recorded.

CASE XXVIII.—Small male infant, aged eight days. No clinical history. The umbilical stump was still adherent. There was interstitial hemorrhage involving the substance of the left suprarenal. The right was soft, cyst-like, enlarged, and on

section showed a cavity the size of a large cherry which was filled with blood. The walls of the cyst were not disorganized. The mesenteric glands were enlarged, brownish and probably hemorrhagic. The stomach contained considerable grumous material having the appearance of altered blood. The cerebral vessels were injected. Cultures made from the spleen and right suprarenal showed staphylococcus aureus and albus.

CASE XXIX.—Male infant aged eight days. No clinical history. The umbilical stump was attached but mummified. The umbilical vein was large, bluish, and occupied in its entire length by a soft clot. The hypogastric veins also contained clots. The visceral pericardium was injected; the lower lobe of the lungs highly congested, and both suprarenal glands were enlarged, congested, and in some areas hemorrhagic. The kidneys were slightly congested.

CASE XXX.—Male infant aged eight days. No clinical history. The autopsy showed thrombosis of the umbilical vein; hemorrhagic infiltration of the lungs; ecchymoses in the mucous membrane of the intestine and stomach, and engorgement of the mesenteric vessels. The left suprarenal was very soft; on section it contained considerable grumous material. The right suprarenal showed a hemorrhage into its medullary substance.

CASE XXXI.—New-born male infant, age not given. No clinical history. The right suprarenal gland was soft on section. It showed no cavity but considerable blood oozed on section. The medullary portion was dark red and hemorrhagic. There was universal hemorrhage of the right kidney, affecting especially the pyramids; the pelvis was injected and bluish-black. The left kidney was enormously enlarged and black, and on section was full of blood. The lungs were slightly congested. The umbilical vein contained a thrombus at its umbilical end. The inferior cava contained clots, and one extended to the right renal vein. It was dark in color and quite firm. Cultures were made from the left suprarenal.

Dr. Riesman tells me that in the three cases in which cultures were made and the results not recorded, the staphylococcus albus and aureus were found in each.

Mattei (*Lo Sperimentale*, 1863, p. 28, and *Jour. de Med. de Chirurg. et de Pharmacol. de Bruxelles*, 1865, Vol. XLI, p. 327), also observed five cases of hemorrhage into the suprarenal

gland in new-born infants dying before the sloughing of the cord. Two of these cases are reported in detail.

CASE XXXII.—Born at term; well developed; died on the second or third day. The suprarenal glands were enlarged; chestnut-colored; the parenchyma infiltrated with blood, giving a deep red color on section. The large capsular vein was turgescent. There was infiltration of blood into the elastic tissues about the gland. There were many areas of slight hemorrhage under the pia mater and on the surface of the cerebral hemispheres.

CASE XXXIII.—Four-day-old infant. No forceps lesion; subpleural ecchymoses, large hemorrhage into the left suprarenal gland which ruptured into the abdominal cavity. The parenchyma of the gland was entirely infiltrated with blood. On section there were two pockets visible which had contained the blood that had escaped into the abdominal cavity. The intraabdominal clot extended into the pelvis and surrounded the left kidney and the lower part of the left suprarenal gland. The right gland showed a slight infiltration of blood into its medulla. The kidney and liver were pale.

CASES XXXIV, XXXV and XXXVI are referred to by Mattei above but not described.

Mattei (*Lo Sperimentale*, 1863, p. 28, and *Jour. de Med. de Chirurg. et de Pharmacol. de Bruxelles*, 1865, Vol. XLI, p. 327) observed ten instances of hemorrhage into the suprarenal capsule in autopsies on twenty-two fetuses. He reports five of these in detail, as follows:

CASE XXXVII.—The author observed apoplexy of both suprarenal glands in a fetus born dead at full term. The glands were much larger than normal, but preserved their normal form. Underneath the capsule were various spots of extravasated blood, and on section they showed a deep red color uniformly diffused. Fluid blood exuded freely from the cut surface on slight pressure. The condition was an interstitial apoplexy, the blood probably coming from the rupture of a considerable number of small vessels. The liver was enlarged and engorged with blood. The other abdominal organs were normal.

CASE XXXVIII.—Nine months' fetus; well-formed; large. The head was engaged many hours in the lower pelvis, during which time the fetus died. It was extracted by cephalotripsy. The cord was once around the neck. This was considered to

have caused death when the head descended into the pelvis. The suprarenal glands were a little enlarged and contained many small cavities filled with fluid blood. The walls of the right cardiac ventricle were hypertrophied; the other organs were normal.

CASE XXXIX.—Male; born at term; well developed. The lungs contained air. The suprarenal glands were large; the parenchyma of both was a deep red and showed hemorrhagic infiltration. Under the capsule of the right, over more than one-half of its anterior surface, there was a thin stratum of blood; the same gland contained a small cavity filled with liquid blood. There was a considerable amount of blood over the posterior half of the cranium, and a thin stratum overlying the upper and under surface of the cerebellum. There were many small ecchymoses over the lungs; the other organs were healthy.

CASE XL.—Fetus delivered by cephalotripsy many hours after the membranes had ruptured. There was hemorrhage into the substance of the right suprarenal gland under its posterior angle. The left was healthy. There were small ecchymoses in the visceral pericardium. All the other organs were normal.

CASE XLI.—The suprarenal glands were a little enlarged with small ecchymoses over their surfaces. The parenchyma of each was dark red and infiltrated with blood. Under the scalp in the occipital region, as well as over the arachnoid and superior surface of the cerebellum, there were small extravasations of blood. The cerebellum was softened in many points.

CASES XLII to XLVI, inclusive, are referred to above by Mattei but are not described.

Cases observed by Spencer (*Obstetrical Transactions*, London, Vol. XXXIII, 1891) in still-born children.

CASE XLVII.—Female; both suprarenals distended with blood clots; the left ruptured, and the blood was spread behind the kidney. Both kidneys were hemorrhagic and the liver congested. There was hemorrhage over the vertex of the skull and under the periosteum of the left frontal and the right occipital lobes. The meningeal vessels were congested.

CASE XLVIII.—Female; accidental hemorrhage; version. Hemorrhage into the medulla of the suprarenals; into the cellular tissue of the scalp; liver, lungs and meninges congested.

CASE XLIX.—Female; hydrocephalus; breech presentation; traction; suprapubic pressure. The right suprarenal capsule was ruptured, the left congested. The right lobe of the liver was ruptured at its posterior part; ecchymotic areas in the lungs and heart; subcapsular hemorrhage into the right kidney; both congested; hemorrhage into the peritoneum and the tissues of the labium majora.

CASE L.—Female; breech presentation; easy delivery. The left suprarenal was greatly congested and there was hemorrhage into its medulla. There were subpericardial and pulmonary ecchymoses, with congestion of the peritoneum, small intestine, rectum, left kidney, uterus, ovaries and cerebral vessels.

CASE LI.—Male; slight hemorrhage into the medulla and congestion of both suprarenals; hemorrhage into the arm muscles, liver (ruptured), base of brain and processus vaginalis.

CASE LII.—Male; contracted pelvis; induced labor; forceps; *child lived two days.* The suprarenals were congested, the walls of the left separated by blood; small hemorrhage and congestion of the intestines and mediastinum testis. Hemorrhage into the tissues of the scalp, on the surface of the right cerebrum and around the spinal cord.

CASE LIII.—Male. Child had imperforate anus and dilated descending colon. The girth of the abdomen was fourteen and one-half inches; delivery natural, vertex. The cord was wound around the neck; hemorrhage into the left suprarenal; great congestion of the right; much congestion of the mediastinum testis, also surface of the testes; hemorrhage and congestion of both kidneys; congestion of brain and pancreas; nails and mucous membranes blue.

CASE LIV.—Female. Contracted pelvis; footling presentation; depression of right parietal bone; traction; occiput rotated backwards. Hemorrhage into medullæ of both suprarenals, in the left it exists as isolated patches, the right is converted into a cyst-like capsule filled with fluid blood; hemorrhage into the mucous membrane of the uterus; into the hilum of both kidneys; into the left lung; into the scalp; on the surface of the brain; into the anterior cornua of the lumbar region. Both kidneys congested.

CASE LV.—Male. Natural vertex. Hemorrhage between suprarenals and kidneys; much hemorrhage into the suprarenals; hemorrhage behind both kidneys and into the cellular

tissue of the hilum of the kidneys; scrotum, right spermatic cord, mediastinum testis, lungs and spinal cord congested. Hemorrhage under the pericranium and over the surface of the temporosphenoidal lobes.

CASE LVI.—Male. Placenta previa; version; embryotomy for severe hemorrhage in mother. Suprarenals were full of blood; hemorrhage into the liver; kidneys, testes and lungs congested.

CASE LVII.—Male. Cephalotripsy. Head hard and well ossified. Suprarenals large; the left has its lower half distended with blood; hemorrhage into the spinal arachnoid; liver and kidneys congested.

CASE LVIII.—Male. Mother secundipara aged thirty-four; last child seven years previous; flat pelvis; slight hydrocephalus; forceps; two convulsions; version; strong traction; child just alive when born. Right suprarenal covered for a space of one and one-half by one and one-eighth inches by a layer of black blood which escaped through a laceration in the capsule and its peritoneal investment; congestion of the testes, lungs, spleen, cerebellum and medulla; hemorrhage into the thymus, both lungs, scalp, right Sylvian fissure, and over both temporosphenoidal lobes.

CASE LIX.—Male. Natural vertex presentation. Child died in convulsions *three hours after birth.* Suprarenals distended with fluid blood; slight superficial hemorrhages on surface of pulmonary artery, beneath pericranium; head and face, liver, spleen and kidneys congested.

CASE LX.—Female. Mother had epileptic fits for two days before delivery; version followed by natural delivery fourteen hours later through a rather rigid cervix. Suprarenals much congested and showed slight hemorrhage. Hemorrhage over left hemisphere, at base of brain, and over left cerebellum.

CASE LXI.—Female. Mother multipara; labor twelve hours; *child died eighteen hours after birth* of septicemia contracted in utero; vertex. Suprarenals full of bloody fluid; skin bluish; bloody fluid in pericardium, peritoneum, pleuræ and arachnoid; hemorrhage into hilum of kidneys; into cellular tissue around uterus and ovaries, and into all the subperitoneal and cellular tissues. Lower left lung congested.

CASE LXII.—Male. Contracted pelvis; forceps. Hemorrhage in front of suprarenals, also into substance of organ, and

in cellular tissue between suprarenal and kidney. Hemorrhage into the scalp; subperiosteal hemorrhage over both parietal and over both frontal bones; slight at the base of the cerebellum and around the medulla, over the nose and petechiæ over lungs; kidneys congested.

CASE LXIII.—Male. Accidental hemorrhage; second breech presentation; legs extended; forceps; traction by fillet and groin. Hemorrhage into left suprarenal; right slightly congested; bruise in left groin from traction; small quantity of blood in peritoneum and in each tunica vaginalis; hemorrhage into mediastinum testis, liver, subpericardium, periosteum, over left side of surface and at base of brain and into meninges of spinal cord; left kidney cortex greatly congested; lungs congested.

CASE LXIV.—Female. Multipara. Rigid cervix; accidental hemorrhage; footling presentation; strong traction. Hemorrhage into right suprarenal; legs black; black bruise on left shoulder and on back. Hemorrhage into left sternomastoid, temporal muscle, gluteus maximus, erector spinæ, cellular tissues and muscles of legs and cellular tissues of right thigh. Hemorrhage into scalp, pleuræ, lung, liver, and rupture of its capsule; into great omentum; into hilum of both kidneys; on the surface of the left cerebral hemisphere at the base of the brain and between the dura and arachnoid; spinal cord congested.

CASE LXV.—Female. Labor eight hours; membranes prematurely ruptured; breech presentation; delivery natural until shoulders were born, when child gasped and was delivered with difficulty by the midwife, still-born. Slight hemorrhage into the medulla of both suprarenals at upper part; vessels of uterus and Fallopian tubes congested; hemorrhage on the surface of brain.

CASE LXVI.—Male. Eighth month. Mother multipara aged thirty-nine; five-hour labor; breech; arms extended; extraction of head difficult; *heart beat for twenty minutes* but the child never breathed. Both suprarenals congested; slight hemorrhage into the left; hemorrhage into cellular tissue and muscles of back and thigh, and into the lower third of the right sternomastoid. Slight hemorrhages into the cellular tissue just above the periosteum of the scalp; hemorrhage on the surface of the brain; under the capsule of the liver; into testes; dartoid tissue. Kidneys congested.

CASE LXVII.—Male. Natural vertex. Child revived by artificial respiration; *died suddenly fifteen minutes later.* Hemorrhage into suprarenals; into scalp and upper surface of the right lobe of liver; into the capsule of the spleen and into its substance; at the base of the brain and into tentorium cerebelli; hemorrhage and congestion of cellular tissue around the kidneys and in the hilum; testes congested.

CASE LXVIII.—Female. Natural first vertex delivery. Suprarenals congested; slight hemorrhage into left; hemorrhage over surface of liver, subcapsular; into lungs; beneath the visceral pericardium; into the Schneiderian membrane; under the parietal periosteum; over surface of parietal lobes; into duodenum; fulness of the veins of the upper cerebellum; congestion of the mucous membrane of the stomach, esophagus and jejunum; deep ecchymoses in cortex of both kidneys; thymus congested.

CASE LXIX.—Female; breech; extended legs; impaction failed to bring down leg; traction with fillet and groin; arms extended, difficult to bring down; child died during delivery of arms. Suprarenals both congested; hemorrhage into right; hematoma of the left labium minus; hemorrhage into cellular tissue around the orifice of vagina; slight hemorrhage at base of brain; under laminæ; into anterior edge of left sternomastoid; into hilum of kidneys; congestion of thymus and of the pyramids of the kidneys.

CASE LXX.—Portal (cited by Lieutaud and quoted by Droubaix) reported the case of an infant two months old which cried continuously for five days without discoverable cause. Convulsions developed and the child died after three days. The autopsy showed hemorrhage into both suprarenal glands. The glands were larger than a pigeon's egg.

CASE LXXI.—Wainwright (*Trans. of Path. Soc. of London,* 1893, Vol. XLIV, p. 137.) A two-months-old infant with purulent ophthalmia and bronchopneumonia died in convulsions.

Autopsy.—Areas of consolidation were present in both lungs. There were no evidences of tubercle bacilli. All the other organs were healthy except the adrenals; one measured one and one-eighth inches in length by five-eighths of an inch in breadth and one-third of an inch in thickness. It was tough, pale and slightly nodular. On section it showed a cortical layer

of apparently normal tissue bounded internally by a band of brownish pigment; internally to this was a practically translucent caseous looking mass which felt gritty under the knife. Microscopically the outer layer was made up of normal cells with here and there patches of broken down tissue. The band of pigment was the remains of an old hemorrhage. The central portion was composed of a coarse, fibrous, net-like material containing in parts small cells, but chiefly enclosing mulberry-like calcareous masses and a few patches of dark pigment. There were no tubercles, no leucocytic infiltration, in fact, nothing to suggest a recent infiltration. The condition was thought to be an old hemorrhage.

CASE LXXII.—Duckworth (*Twentieth Century Practice*, Vol. II) refers to a case of pertussis in an eight-months-old child in which the paroxysms of cough induced excessive hemorrhage into both adrenals.

CASE LXXIII.—Still (*Trans. of Path. Soc. of London*, 1898, Vol. XLIX) discovered at an autopsy on a fourteen-months-old infant dying of acute miliary tuberculosis a distended purplish suprarenal capsule. On section the whole organ was engorged with blood. The medulla was of a dark purple color. There were one or two gray tubercles present in the substance of the gland. Microscopically the medulla was infiltrated with extravasated blood corpuscles, but there was no disintegration of the tissues. The hemorrhage was nowhere circumscribed. The cortex showed engorgement of the small blood-vessels and some extravasation of blood, but less than in the medulla.

CASE LXXIV.—Andrews (*Trans. of Path. Soc. of London*, 1898, Vol. XLIX, p. 259). Female infant, aged fifteen months, had a hemorrhagic rash which was papular in the chest and somewhat resembled small-pox. It died after an illness lasting two days.

Autopsy.—The glands of the neck were somewhat swollen. All the viscera except the suprarenal glands were normal, both of the latter were dark red in color from hemorrhage. Cultures from the suprarenals, lungs, liver, spleen, and kidneys remained sterile. Blood films from the suprarenals stained in various ways showed no microorganisms. Sections from the suprarenal glands, lungs, liver, kidney and spleen were stained but showed no microorganisms. The suprarenals

showed a firm uniform diffuse extravasation of blood, the proper tissue elements being obscure and apparently diminished in number.

CASE LXXV.—Churton (*Lancet*, 1886, Vol. I, p. 248) reports a case of hemorrhage into both suprarenal capsules in an infant dying as the result of a severe burn.

CASE LXXVI.—Voelcker (Registrar's Reports, Middlesex Hospital, 1894, p. 278) notes the case of an infant dying at the age of two years from an acute illness and having purpura. The autopsy revealed hemorrhage into both suprarenal glands.

CASE LXXVII.—Batten (*Trans. of Path. Soc. of London*, 1898, Vol. XLIX, p. 258.) An infant aged two and one-half years had suffered from urticaria for one month. He was taken suddenly ill with vomiting, diarrhea and fever. Convulsions occurred on the second day. The child became comatose. The respirations were of the Cheyne-Stokes type. The pulse reached 200 beats to the minute. The temperature rose to 102° F. Strabismus was present. There were no fundus changes. There were bronchial rales over the entire chest. The knee jerk was marked and there was a tendency to ankle clonus. The temperature rapidly rose to 106° F., and the child died on the evening of the second day.

Autopsy.—There were three small extravasations of blood on the floor of the lateral ventricles, congestion of the lower lobes of the lungs to a dark purple color, and of the right suprarenal gland. All the other organs were normal. A microscopic examination showed the entire suprarenal to be extremely congested, extravasation of blood having taken place into the medullary portion of the organ. The left suprarenal was normal.

CASE LXXVIII.—Garrod and Drysdale (*Trans. of Path. Soc. of London*, Vol. XLIX, p. 257). A female child aged four years had a blotchy purpuric eruption over the body. The thymus gland was large. Both suprarenals were of a deep purple red color, and their medulla was of a deep purple tint. There were no circumscribed hemorrhages. Microscopic examination showed the stroma fairly well preserved. The cells enclosed in the meshes of the stroma had largely disappeared being replaced by effused blood. Some of the individual spaces were entirely filled by red

blood corpuscles; elsewhere the cells could be seen in considerable numbers, the nuclei staining well, but the cell substance being practically destroyed. There were no other lesions in the body. Cultures from the spleen, liver, kidneys and suprarenal capsule remained sterile.

CASES LXXIX TO XC, INCLUSIVE.—Still (*Trans. of Path. Soc. of London,* 1898, Vol. XLIX), in commenting upon his case described above says that in 3791 autopsies on children under the age of twelve years at the Hospital for Sick Children, Great Ormond Street, only 4 cases of suprarenal hemorrhage were noted. One occurred in a child aged three years and the others in infants. Two cases of marked congestion, one in a child of eleven years dying of acute tuberculosis and ulcerative endocarditis, the other in an infant dying of septicemia were also recorded.

In addition to the above, on autopsies made elsewhere in twenty-five children dying before the fifth day, he found congestion in 4 and hemorrhage in 1 case. He refers to records of 2 additional cases of congestion and 1 of hemorrhage into the suprarenal gland in infants, no particulars being recorded, making a total of 12 cases of hemorrhage and 7 of congestion. These 12 cases with the 78 already recorded bring the total up to 90.

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FOOT NOTE.—Since this article has gone to press it has been brought to my attention that Dr. Chas. Norris presented to the New York Path. Soc. on March 12, 1900, a case of "Hematoma of the Right Suprarenal which Ruptured into the Peritoneum in a Child aged Ten Days." The infant was a blue baby and died suddenly on the tenth day. The only other lesions recorded are "a foramen ovale nearly closed" and "a ductus arteriosus presenting a funnel-shaped opening at its aortic end." He refers to having seen an exactly similar case. In the discussion of this case Dr. Larkin refers to having seen four or five hematomata in still-born children.

THE BLOOD IN INFANCY AND CHILDHOOD.

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THE ERYTHROCYTES.—The red corpuscles are more numerous at birth than in the normal condition in after-life. The average of the enumerations of various investigators is 5,742,080 per c.mm. The counts obtained by these investigators are as follows:

Hayem	averaged	-	-	-	5,360,000
Sörensen	"	-	-	-	5,665,000
Otto	"	-	-	-	6,165,000
Bouchat and Dubrisay	"	-	-	-	4,300,000
Schiff (one case)	"	-	-	-	6,658,000
Gundobin	"	-	-	-	6,700,000
Elder and Hutchinson	"	-	-	-	5,346,560

Schwinge greatest at birth.

Certain attending conditions are supposed to influence the number of cells, thus according to Hayem and Helot it has been found that when the umbilical cord was not tied until its pulsations had ceased, a greater number of red corpuscles was found than in cases in which immediate ligation was practiced.

Elder and Hutchinson in comparing the new-born infant's blood with that of its mother found the former always richer in the number of corpuscles, the difference being as much as 350,000 to 500,000 per c.mm. Gundobin attributed this high count to concentration of the blood by loss of water through the lungs. Schiff found the same and further that the number of corpuscles decreased when the child was put to the breast. Elder and Hutchinson and Gundobin speak of the variations in the daily count, but do not attribute such variation to the usual physiological processes such as the influence of diet, and Gundobin suggests that it may be the result of some change in the

chemical composition of the blood as a whole. The number of red corpuscles begins to fall after the second day and in one case in which Schiff estimated the number in the morning and evening during the first fifteen days of life, there was an irregular declension. The first day's count was 7,628,000; the last day's count was 4,565,600; and the average for the fifteen days was 5,828,465. This decrease in the number continues during the first year according to Schwinge and Gundobin and then there is an increase up to the eighth or twelfth year when the number becomes approximately that maintained until adult life. The count of the two sexes is approximately the same both before the fourteenth or fifteenth years and after the menopause, but in the intervening years the count in women is apt to be lower than that in men.

The red corpuscles during the first few days and at birth vary greatly in size. Hayem estimated the variations at from 3.25 μ. to 10.25 μ. and Loos found the size from 3.3 μ to 10.3 μ. This irregularity in size has been observed by many others. Gundobin claims that the hemoglobin is more firmly attached to the cell stroma in the new-born infant and he also calls attention to the great number of small-sized corpuscles. In general, however, the histologists find no difference in the structure of the red corpuscles in infancy and in the adult.

THE HEMOGLOBIN.—This is increased at birth as Taylor, Morse, Elder and Hutchinson, Rotch, and others have shown, but it tends to decline rapidly in the first few days of life. Gundobin found the proportion of hemoglobin greater at birth than in adults or in infants after feeding had begun. Rieder's investigations showed an excess of 25 per cent. to 30 per cent. at birth.

SPECIFIC GRAVITY.—This usually varies as the percentage of hemoglobin varies, so that at birth the specific gravity is high and subsequently it declines. Monti found it 1060 at birth; Rotch 1065; Hotch and Schlessinger 1066; and Moelle 1060. E. Lloyd Jones noted that the specific gravity was highest at birth and at a minimum between the second week of life and the second year.

It has generally been observed that the specific gravity like the number of cells decreases after the first two days. Hoch and Schlessinger found figures between 1048 and 1052 up to

two years of age and 1052 to 1056 from two to six years. The following figures will indicate the specific gravity at different periods.

Monti found the average	1057	at two to four weeks.
" " " "	1050	" twelve months.
" " " "	1052	" two to ten years.
Rotch " " "	1048-1051	up to two years.

The investigations of Monti, Rotch and Hoch and Schlessinger show that the specific gravity may be stationary for weeks or months at a time in healthy children. The variation, for example, in two healthy children studied by Hoch and Schlessinger was only .0025.

THE LEUCOCYTES.—The white blood corpuscles are greater in number at birth than in the adult blood, this excess in number constituting that which has been recognized as the physiological leukocytosis of the new-born. The following figures have been found by the authors quoted.

Rieder to be	15,500	10 minutes after birth.
Oransky " "	16,980	immediately after birth.
Cadet " "	19,480	" " "
Elder and Hutchinson to be	17,884	average in 12 cases at birth.

During the first forty-eight hours of life there is a still further increase in the number of leucocytes after which the number declines, though the count still remains higher during the first and second years than that found in the blood of the adult. The following table shows figures obtained at various times after birth:

Schiff	24,000-36,000	in first 24 hours.
Oransky	20,980	20 hours.
"	31,680	next day.
Gieffer	18,000	24 hours.
Rieder	16,500	8 hours.
After the third day		
Rieder 1 case	8,700	3d day.
" 2 cases	10,500	5th day.
" 3 cases	13,600	4th day.
" 3 cases	12,200	5th day.

After the second year the number gradually declines to that found in adult blood and the percentage of the various forms of leukocytosis also becomes normal.

Physiological influences, such as diet and digestion, have about the same effect on the leucocytes in the infant as in the adult, that is to say, a digestive leukocytosis is observed. From the frequent feedings of the infant, however, this leukocytosis is practically constant as Taylor has pointed out, but Gundobin has observed an increase of from 2,000 to 4,000 in the number of leucocytes after feeding. The same author has observed that daily variations of temperature have no effect.

With regard to the variety of leucocytes it may be noted that the same kinds of cells are found as in the adult blood though the proportions of the several forms are different. The most striking peculiarity in the differential count is the increase in the number of lymphocytes and the more or less proportionate decrease in the polymorphonuclear cells.

Gundobin gives the following figures: Lymphocytes 50 per cent. to 66 per cent., polymorphonuclear 28 per cent. to 40 per cent. This indicates a three-fold proportion in the number of lymphocytes as compared with the adult, and a corresponding paucity of the polymorphonuclear cells amounting to about a half. The weight of the child apparently has no influence either on the total number of leucocytes or on the proportions of the different forms. If the child is increasing normally in weight, the numbers already alluded to occur, but when there is a cessation of the normal growth or a decrease in weight, variations in the number of leucocytes and in the relative proportions of the various types are apt to appear. Daily variations of temperature or artificial elevations of temperature amounting to 0.6 per cent. C. apparently have no influence on the number of leucocytes. (Gundobin.) C. S. Engel found 12 per cent. to 20 per cent. of polymorphonuclear cells in infants during the first few months of life and 40 per cent. to 50 per cent. after the expiration of the first few months up to the end of the first year. At twelve years of age he found 60 per cent. of polymorphonuclear cells.

The eosinophile cells vary greatly in number at birth and we find expressions as follows: "almost fail" (Elder and Hutchinson); "not increased" (Weiss); "in varying numbers" Loos); "1.53 per cent. to 19.54 per cent." (Zappert); "often considerably increased" (Hoch and Schlessinger).

Nucleated red corpuscles are the embryonal type of the normal erythrocyte and, until the sixth month of intrauterine

life, form the greatest number of the red cells of the blood. From this period until birth they gradually decline in number and at birth only a few erythroblasts can be found. By the end of the second day these as a rule disappear entirely. A few observers like Hoch and Schlessinger have found them in apparently healthy children. As a rule they are not found after the second or third day, excepting in children who are ill. Elder and Hutchinson found them as numerous as 1 to 20 and 1 to 8 of the leucocytes, in the blood taken from the umbilical cord. They also noted many free nuclei but no mytoses.

PATHOLOGICAL CONDITIONS OF THE BLOOD IN INFANCY.

The first changes observed in most cases of disease affecting the blood is a reduction in the hemoglobin and in the number of the erythrocytes, but as a rule the reduction in coloring matter is greater than that of the number of cells, especially in young infants and early childhood. No other change may be observed in the red cells, but on the other hand, there are often changes in size and shape. In the blood of infants Gundobin calls attention to the occurrence of the smaller forms, the so-called microcytes. A condition of the red corpuscles that has been commonly regarded as degenerative is its peculiar reaction to stains as a result of which the red cell becomes dichromatophilic or polychromatophilic. Loos has called attention to the microcytes and macrocytes as showing this change in particular, and the same author calls attention to the fact that the biconcavities of the same cells are usually lost, showing a change in the structure as well as in the staining properties of the cells. A still further change in these cells causes an increased adhesiveness so that they stick to the cover glass firmly. Loos' attention was first directed to this by observing that in specimens insufficiently fixed these cells and the leucocytes were the only ones to remain after washing the slides for mounting. The polychromatophilic change is a rather common one, being found in posthemorrhagic conditions and in most of the anemias. There is much difference of opinion as to the real cause or actual change in the cell. Ehrlich supposed that it was an evidence of senility or of death of the cells while Gabritschewski, Askanazy, Dunin and others opposed this theory and state that they have found it in young cells, *e.g.* around the nucleus of the megaloblasts. Basic or

granular degeneration of the red corpuscles has not received the attention of investigators in the blood of children. Loos found cells with fine granules which he supposed to be the remains of a former nucleus and Ehrlich suggests that this change is a coagulation necrosis of the cell contents or that the granules are the remains of a former nucleus.

Nucleated red corpuscles: Erythroblasts. When these cells are found in the circulating blood after the second or third day of life, their presence may usually be assumed to indicate a pathological change or condition. The significance of erythroblasts is, however, much less in infancy and childhood than in the adult since marked anemia occurs so much more readily in early life. The number of the erythroblasts varies greatly at different times in the same patient and in like grades of intensity of a similar disorder in different patients. Erythroblasts have been found in secondary and in primary anemias by Weis, Gundobin, Elder and Hutchinson, Morse, C. S. Engel, Monti, Berggrun, Hoch and Schlessinger. Loos found them very abundant in pseudoleukemia, syphilis, rickets, osteomyelitis, congenital rickets and tuberculosis, and in less number in the same affections in slighter grades. Most investigators have found these cells under similar conditions. Karyokinetic figures are rarely found in the circulating blood and only in cases of very severe anemia.

Leucocytes. Increase in the number of leucocytes (leukocytosis) more frequently presents itself in the blood of anemic children than in adults and the increase may reach enormous proportions in apparently slight pathological conditions. The causes of leukocytosis in childhood are in general the same as those which occasion the condition in adults. We may distinguish toxic, inflammatory, posthemorrhagic, and cachetic forms. Enlargement of the spleen may or may not accompany the leukocytosis; in the majority of cases there is enlargement. The differential count of the leucocytes shows an increase in the lymphocytes, the mononuclear cells or the polymorphonuclear neutrophiles. The eosinophiles seem to be governed by influences quite different from those which control the number of the other forms, but the nature of those influences is as yet unknown. Myelocytes are more frequently found in the blood in childhood than in adults. Their occurrence in increasing numbers is of

bad prognostic significance as C. S. Engel, Cabot, and others have especially noted in pneumonia and diphtheria.

We may now proceed to the consideration of the hematologic features of various general and local diseases.

INFECTIOUS DISEASES.—During the attack there is often but little change in the number of red corpuscles and the percentage of hemoglobin, while during convalescence a moderate or severe grade of anemia presents itself. This is explained by the assumption that the blood is inspissated in the febrile stage of the disease by increased action of the skin and lungs or by diarrhea. When anemia develops, the reduction in hemoglobin usually exceeds that of the number of corpuscles, and at times this disproportion is marked. The number of leucocytes differs in various diseases, being increased in some, and unaffected or decreased in others. Among those in which a decrease (or at least no leukocytosis) is observed, are rötheln, variola in its earlier stages (Pick), mumps (Cabot), influenza, malaria, typhoid fever, tuberculosis (before excavation and in miliary tuberculosis), and varicella (Loos and Engel). Very slight causes may determine a moderate leukocytosis in any of these as in healthy children. In other infectious processes and in those above named when complicated by inflammatory or other conditions considerable and often excessive leukocytosis may be met with.

With regard to the variety of cells mainly involved in such infectious leukocytosis, Weiss and Gundobin found the polymorphonuclear cells especially increased in diphtheria, scarlatina, erysipelas and pneumonia. Gundobin in addition found that the increase of the leucocytes occurred some time before the eruption in scarlet fever, measles and erysipelas. C. S. Engel found 67 per cent. of polymorphonuclear elements and no eosinophiles in varicella and three days later, when the skin lesions had healed, only 47 per cent. of polymorphonuclear cells and as many as 16 per cent. of eosinophiles. In measles Weiss failed to find increase of eosinophiles. In typhoid fever the number of leucocytes is decreased in childhood as in adults, and there may be at the same time a decrease of the hemoglobin and the red corpuscles. The number of leucocytes is relatively increased or properly speaking the polymorphonuclear elements are the ones actually deficient, the mononuclear cells, large and small, being present in about the normal number.

The hematological conditions in *pneumonia* are particularly interesting. There is nearly always some degree of leukocytosis and often excessive grades. When the condition is absent, the prognosis is unfavorable in childhood as in adults. Gundobin found in six cases an average leucocytic count of 24,300 with the following differential count: lymphocytes 25 per cent., mononuclear, 6 per cent. polymorphonuclear 70 per cent., eosinophiles 2.5 per cent. Engel found the polymorphonuclear cells excessive and the eosinophiles wanting during the febrile period of the disease, the latter forms reappearing after the crisis. Rotch found that leukocytosis develops at from six to twelve hours before physical signs are discoverable, and that a leucocytic crisis may antedate the crisis of temperature by twenty-four hours. A blood lysis is, however, more common.

In *hereditary syphilis* there is a more or less pronounced grade of anemia according to the severity of the symptoms. Usually the anemia is quite marked. In a careful study of the subject, in which Gundobin, Weiss, Monti and Berggrun, Bieganski and Engel are quoted, Loos arrives at the following conclusions:

1. Hereditary syphilis is accompanied by an anemia which under certain conditions may become very great.

2. The anemia is characterized by a decrease of erythrocytes with great degenerative changes (poikilocytosis) and especially the occurrence of microcytes and macrocytes, by the presence of polychromatophilia and nucleated erythrocytes which may be very numerous at times.

3. A leukocytosis at times reaching very high grades and showing a predominence of the small lymphocytes is usually seen.

4. The presence of myelocytes is noted.

5. Hemoglobin greatly decreased.

These changes indicate nothing that may be regarded as peculiar to hereditary syphilis as Rotch and Weiss have pointed out.

Engel found in 15 cases a low percentage of polymorphonuclear forms, 16 per cent. to 11 per cent., while the lymphocytes were considerably increased, and as many as 14 per cent. of eosinophiles occurred in some cases. Nucleated red corpuscles were found in some cases. Loos found myelocytes in 4 cases. Gundobin found the lymphocytes absolutely and

relatively increased. This predominence of lymphocytes over the polymorphonuclears may disappear when malnutrition or complications in the gastrointestinal tract and lung occur.

DISEASES OF THE RESPIRATORY TRACT.—Slight acute inflammatory processes of the respiratory tract may cause oligochromemia and oligocythemia; chronic processes scarcely ever do. The leucocytes generally increase in number according to the severity of the process; slight leukocytosis occurring in the acute catarrhal processes and enormous increase in number when more tissue is involved and the severity of the process is pronounced.

In conditions producing cyanosis there may be increase in the number of red and white corpuscles and in the percentage of hemoglobin. Grawitz found such to be the case in asthma and heart diseases. The polymorphonuclear cells usually contribute the leucocytic increase; the eosinophiles are either not affected or absent altogether in the majority of processes, excepting in asthma of the bronchial type when they are generally spoken of as prominent. Schreiber in his lectures claims that they are not peculiar to this type but are found in all types in the blood and secretions.

In bronchitis there may be slight leukocytosis with especial increase of the lymphocytes or mononuclear cells. The average count and differential count of cases reported by Gundobin is: total 17,500; lymphocytes 42 per cent., mononuclear 8 per cent., polymorphonuclear 50 per cent., and eosinophiles 2 per cent. Weiss found in cases not specially classified as acute or chronic moderate leukocytosis with particular increase of the mononuclear forms. In one of the cases in which nasal complications existed he found an increase of the eosinophiles.

GASTROINTESTINAL DISEASES.—The condition of the blood varies according to the extent of the process the duration, and the existence or non-existence of diarrhea and vomiting. Profuse diarrhea or vomiting may for a time thicken the blood by loss of water. Hoch and Schlessinger found that such inspissation with consequent increase of specific gravity does not occur until the drain has existed for some time and the tissues as well as the blood have been affected. There is no change in the blood, according to their investigations, when the amount of water lost is equalled by the quantity ingested. When the ingestion

is less than the excretion the tissues first contribute and the blood secondarily.

The differential count of the leucocytes, according to Weiss, shows an especial increase of the lymphocytes and transitional leucocytes:

RACHITIS.—In rickets there is no typical blood-picture. The changes found vary with the severity of the affection, its duration, and the involvement of the inner organs. In the moderate grades there is usually a reduction of red corpuscles and a decrease of the hemoglobin with an accompanying leukocytosis. In severe cases these conditions become pronounced. Weiss found increase of the mononuclear forms and transitional leucocytes. The neutrophiles were decreased. Loos found nucleated reds, myelocytes, polychromatophilia. The red corpuscles in some cases show enormous reductions in a comparatively short space of time (v. Jaksch, Luzet.)

CUTANEOUS DISEASES.—Increase in the number of eosinophiles has been observed in a variety of skin diseases, but the cause of the increase is entirely unknown.

NERVOUS DISEASE.—In the functional disorders of childhood there may be a moderate grade of anemia, though this is often less marked than the appearance of the skin would indicate. Burr has found that the blood in chorea is not as a rule anemic.

METHOD OF EXAMINATION.

In our own examinations of the blood in childhood, we have enumerated the corpuscles and estimated the hemoglobin in all cases and have made differential counts of the leucocytes. In some cases we have estimated the specific gravity of the blood, but have not pursued this as a routine. In the histological examination of the blood, various methods of preparation were used; the films were fixed with heat, mixtures of absolute alcohol and ether, solutions of bichlorid of mercury, picric acid, etc. These various methods were employed because we were particularly desirous of discovering any signs of nuclear change which might escape detection by the ordinary method of fixation with heat. The stains used were eosin and hematoxylon, Canon's stain, and Ehrlich's triple stain.

Our examinations have shown practically no differences in the morphology of the red corpuscles in childhood as compared

with the adult. Polychromatophilia and irregularities of shape and size of the red cells were perhaps more conspicuous in the moderate anemias than in the adult and nucleated red corpuscles were found in some cases in which the degree of anemia would not have led us to expect their occurrence in adults. In no case was granular basic degeneration observed.

In the study of the white corpuscles certain peculiarities were observed that merit especial mention. There was a decidedly greater tendency to basic staining than we had been accustomed to observe in adult blood. The lymphocytes stained with Canon's mixture in many cases presented a coarse granular protoplasm while the nucleus stained a light blue. Occasionally this granular character assumed the appearance of distinct granulations and in some instances these granulations were extruded from the cell, projecting as little knob-like masses. These of course suggested artefacts, but if so the occurrence of the coarse granular bodies in the protoplasm indicated that there was before the extension a differentiated condition of the protoplasm and that the extruded particles represented performed elements and not artefacts pure and simple. In the large mononuclear cells we found in a number of instances minute basic or amphophilic granules. Even when amphophilic these rather inclined to basic than acid affinity. These granules were closely set and gave the protoplasm of the cell a fine dusted appearance. Coarse basophilic granules were occasionally found in these cells, but never distinct mast-cell granules. The polymorphonuclear cells in a few instances contained very sharply defined and quite abundant basophilic granules. These were larger than the neutrophile granules usually observed, but were smaller than mast-cell granules. They stained with great intensity and therefore gave the cell a very striking appearance. Mast-cells were found in some cases but were not abundant in any instance. Upon the whole, the basophilic granules were much more conspicuous in the blood of childhood than they have usually been found or we have found them in the blood of adults.

Myelocytes were observed in a number of cases (10 out of a total 49) as will be seen in the special notes. We could find no particular significance in their occurrence. In connection with what has been said before regarding the prognostic significance of myelocytes, it may be noted that one of the most

severe cases of pneumonia ending fatally showed as many as 2.2 per cent. of myelocytes at one of the examinations.

The blood counts in general have shown no striking peculiarities. The number of leucocytes was usually high as compared with the numbers found in adults excepting in the case of typhoid fever in which the leucopenia usually observed was found. Transitional leucocytes were estimated with the large mononuclears.

We may now refer to the cases in detail and summarize the observations after the histories of the cases.

PNEUMONIA.

CASE I.—Helen D., aged nine years, was admitted to the hospital with croupous pneumonia. There is nothing of interest in the clinical history which was incomplete.

The blood count showed: 4,460,000 red blood corpuscles; 29,200 white blood corpuscles; 85 per cent. of hemoglobin. The differential count showed: 87 per cent. polymorphonuclears, 9.4 per cent. mononuclears, 3.6 per cent. lymphocytes, 0.4 per cent. myelocytes.

The following notes were made regarding the appearance of the stained specimens: Canon stain—protoplasm of the leucocytes not well stained but nuclei dark; mononuclears and lymphocytes sometimes difficult to differentiate. Specimens fixed with heat showed some polychromatophilia. Triple stain —distinct myelocytes were discovered.

CASE II.—Lazer T., aged two years and three months. The patient was admitted with pneumonia of the right side, and there were râles throughout the other lung as well. The child was rachitic and the temperature was constantly high. The spleen and liver were both easily palpable, and the former considerably enlarged. Purpuric spots developed on the abdomen. The abdomen subsequently became swollen by tympany. After an illness of some weeks, the child died. No autopsy.

The blood count soon after admission showed: 4,332,000 red blood corpuscles; 68,000 white blood corpuscles; and 56 per cent. of hemoglobin. The differential count showed: 59.6 per cent. polymorphonuclears, 6.4 per cent. mononuclears, 30.4 per cent. lymphocytes, 1.4 per cent. eosinophiles, 2.2 per cent. myelocytes. The second examination, some days later, showed 87,200 white blood corpuscles and 55 per cent. of hemoglobin.

The differential count was then 49.6 per cent. of polymorphonuclears, 8.8 per cent. mononuclears; 39.6 per cent. lymphocytes, 1.4 per cent. eosinophiles (polymorphonuclear), 0.1 per cent. mononuclear eosinophiles, 0.5 per cent. myelocytes. The last examination made towards the end of the illness showed: 3,440,000 red blood corpuscles, 27,824 white blood corpuscles; 46 per cent. of hemoglobin. The differential count showed 47.5 per cent. polymorphonuclears, 13.9 per cent. mononuclears, 37.4 per cent. lymphocytes, 0.8 per cent. eosinophiles, 0.4 per cent. myelocytes.

Examination of the stained specimens showed as follows: triple stain—red blood corpuscles irregular in shape; some large flabby forms; some polychromatophilia; large and small nucleated reds with clover leaf multipartite nuclei. No karyokinesis. Picric acid specimens stained with eosin and hematoxylon showed about the same conditions, and one nucleated corpuscle with a karyokinetic figure. Canon stain (fixed by alcohol and ether)—same conditions of red corpuscles and leucocytes; some of the polymorphonuclear forms contain sparse basophilic granules which stand out very distinctly; some of the lymphocytes show the same granules, the latter were decidedly more coarse than the δ-granules of Ehrlich. Practically, the same conditions were found in the specimens fixed by heat, and in those fixed with bichlorid of mercury.

CASE III.—Sarah M., aged four years, had had measles which was followed by a loose cough and dulness of the left lung. The first examination of the blood showed: 4,540,000 red blood corpuscles; 35,200 white blood corpuscles; 85 per cent. of hemoglobin. The differential count showed 71.6 per cent. polymorphonuclears; 10.5 per cent. mononuclears; 17.9 per cent. lymphocytes. The second examination, ten days later, when the child was convalescing, showed: 4,312,500 red blood corpuscles; 18,600 white blood corpuscles; and 90 per cent. of hemoglobin. The differential count showed: 70.5 per cent. of polymorphonuclears, 13.3 per cent. mononuclears, 16.2 per cent. lymphocytes, and 0.2 per cent. myelocytes.

The examination of the stained specimens at the first examination showed slight polychromatophilia, but nothing else of consequence. At the second examination, some poikilocytosis and a few shadow corpuscles were discovered.

CASE IV.—William M., aged four and a half years, was admitted to the hospital with double lobar pneumonia, and developed pericarditis and acute general peritonitis. The examination of the blood soon after admission showed 5,025,000 red blood corpuscles; 34,688 white blood corpuscles; 78 per cent. hemoglobin. The differential count showed 86.1 per cent. polymorphonuclears, 6.2 per cent. mononuclears, 7.6 per cent. lymphocytes, and 0.1 myelocytes.

The microscopic examination showed as follows: Canon stain (fixed by heat)—red corpuscles irregular, some shadow forms, and some polychromatophilia; no nucleated forms; occasional distinct coarse granules in mononuclears and lymphocytes; transition between the polymorphonuclear and the ordinary transitional leucocytes less sharply defined than in normal blood; myelocytes very large. Specimens fixed with picric acid, bichlorid and alcohol and ether showed nothing additional. In the specimens stained with the triple stain, typical myelocytes were found.

CASE V.—Annie S., aged eighteen months. Tuberculous history in mother. Child's illness began with a convulsion, followed by repeated convulsions. No retraction of head. Croupous pneumonia developed. Doubtful meningitis. No autopsy. The blood count showed: 4,962,500 red blood corpuscles; 32,160 white blood corpuscles; and 70 per cent. of hemoglobin. The differential count showed: 84.3 per cent. polymorphonuclears, 8.1 per cent. mononuclears, 7.6 per cent. lymphocytes.

The microscopical examination showed: Canon stain—red corpuscles normal in appearance, though the central parts stain unusually little; mononuclear leucocytes were of two kinds—(1) some with large pale nucleus and slightly granular protoplasm, (2) others of smaller size with dense granulation. A few excessively large, but otherwise typical mononuclear cells were seen; the lymphocytes frequently had a granular zone about the nucleus. Specimens stained with other methods showed nothing additional.

CASE VI.—Josephine G., aged about six years, was admitted with pneumonia of the right lung and had an axillary abscess on the left side. The blood count showed: 3,386,000 red blood corpuscles; 20,400 white blood corpuscles; 69 per cent. of

hemoglobin. The differential count showed: 52.6 per cent. polymorphonuclears, 15.9 per cent. mononuclears, 31.5 per cent. lymphocytes, and there were one thousand nucleated red corpuscles per cubic millimeter.

The microscopical examination showed: Canon stain—slight poikilocytosis; distinct nucleated red corpuscles with clover leaf and multipartite nuclei. Many of the nucleated cells showed polychromatophilia. Mononuclear leucocytes often had distinctly basophilic granular protoplasm. Nothing additional was discovered by other methods.

CASE VII.—Jacob H., aged five and a half years, had had pneumonia at two years of age and was weakly. His present attack began a week before admission. The child was rachitic, and there was a mucopurulent discharge from the nose. Croupous pneumonia of the right apex was discovered. The blood count showed: 3,506,200 red blood corpuscles; 50,917 white blood corpuscles; and 83 per cent. hemoglobin. The differential count was 73 per cent. polymorphonuclears, 11.3 per cent. mononuclears, 14.7 per cent. lymphocytes, 0.9 per cent. eosinophiles and 0.1 myelocytes.

The stained specimens showed: Canon stain—red corpuscles somewhat irregular in shape, but not definitely altered; mononuclear leucocytes slightly granular; some quite large forms with indefinite pale nucleus, probably myelocytes; granular ring surrounded nucleus of lymphocytes; eosinophiles contain very small granules. Nothing additional in the other methods of staining.

In the seven cases myelocytes were found in five though usually in small numbers. In Case II. the percentage reached 2.2 per cent., but this case occurred in a rachitic child and there was besides a hemorrhagic tendency that may have been due to other causes than the pneumonia. Nucleated red corpuscles were found in two of the cases aged respectively two and a quarter and six years. In the former the anemia was marked but in the latter in which the number of erythroblasts was very considerable the anemia was not pronounced. The discovery of a nucleated red corpuscle showing karyokinesis in the one case was noteworthy, though no special significance can be given to this fact. In these cases eosinophile cells were found during the course of the disease when they were discovered at all, but in five of the cases none were found at any stage.

TYPHOID FEVER.

CASE I.—Selina N. P., aged eight years, was admitted with typhoid fever. In addition there was intense bronchitis, with occasional blood-tinged expectoration. This may have been due to bleeding in the mouth. The history is incomplete and the diagnosis somewhat in doubt. Examination of the blood showed: 4,122,500 red blood corpuscles; 27,636 white blood corpuscles; and 77 per cent. of hemoglobin. The differential count showed: 78 per cent. polymorphonuclears, 13.5 per cent. mononuclears, 8.5 per cent. lymphocytes.

The microscopic examination: Canon stain—red and white corpuscles normal in appearance. Some of the lymphocytes present dark basophilic granulation of protoplasm. A few of the red corpuscles are of unusual size.

CASE II.—Theresa K., aged twelve years. The blood count showed: 5,025,000 red blood corpuscles; 6,966 white blood corpuscles, and 77 per cent. of hemoglobin, before a tub bath. The leucocytes counted after a tub bath numbered 13,066. The differential count of the specimen, before the bath, showed: 85.7 per cent. polymorphonuclears; 8.1 per cent. mononuclears: 6 per cent. lymphocytes and 0.2 per cent. myelocytes. After the tub bath there was an increase of the polymorphonuclear leucocytes.

The microscopic examination showed great irregularity in the quality and in the size of the polymorphonuclear cells, as well as in the number and distinctness of the granules. A few definite myelocytes, and several cells of doubtful classification, but probably myelocytes were found. The red corpuscles were rather irregular in shape.

CASE III.—Rachel H., aged twelve years, was admitted in a relapse of typhoid fever which proved of short duration. The blood count showed: 3,320,000 red blood corpuscles; 6,948 white blood corpuscles; and 78 per cent. hemoglobin. The differential count: 55.3 per cent. polymorphonuclears; 9.4 per cent. mononuclears; 34.3 per cent. lymphocytes; 0.9 per cent. eosinophiles.

The microscopic examination showed great irregularity in the size and depth of color of the red corpuscles. Numerous poikilocytes were found, but no nucleated forms or polychromatophilia.

CASE IV.—Ambrose L., aged eleven years, was admitted in the early stages of typhoid fever. Four days after admission, there was some pain in the region of the heart and a rough sound suggesting pericarditis, was discovered. It disappeared, however, in two or three days without effusion. The blood count upon admission showed: 4,565,000 red blood corpuscles; 4,207 white blood corpuscles; and 70 per cent. hemoglobin. The differential count was: 75.4 per cent. polymorphonuclears; 11.6 per cent. mononuclears; 12.4 per cent. lymphocytes; 0.6 per cent. myelocytes. Two hours later after a tub bath there were found: 3,800 white blood corpuscles, and the differential count was 78.7 per cent. polymorphonuclears; 9.5 per cent. mononuclears; 10.2 per cent. lymphocytes; 0.8 per cent. eosinophiles; 0.8 per cent. myelocytes. The examination of the blood during convalescence showed: 4,830,000 red blood corpuscles; 12,320 white blood corpuscles; 80 per cent. hemoglobin. The differential count: 66.5 per cent. polymorphonuclears; 11.7 per cent. mononuclears; 21.6 per cent. lymphocytes; 0.2 per cent. eosinophiles.

The microscopic examination: the red corpuscles were somewhat distorted, and showed a tendency to polychromatophilia, though this was not marked. The lymphocytes were very small and with excessively dark nuclei. A few myelocytes were found and one of these was of excessive size.

CASE V.—Bessie J., was admitted rather late in the course of typhoid fever. The blood count showed: 3,716,000 red blood corpuscles; 6,880 white blood corpuscles; and 70 per cent. of hemoglobin. The differential count showed: 36.3 per cent. polymorphonuclears; 27.3 per cent. mononuclears; 35.8 per cent. lymphocytes; 0.6 per cent. eosinophiles. A short relapse occurred, and the blood count during this (and after a tub bath) showed: 3,850,000 red blood corpuscles; 9,840 white blood corpuscles; and 70 per cent. hemoglobin.

The microscopic examination of the stained specimens showed: Canon stain—a tendency to basophilic protoplasm in the mononuclear cells. There were two mononuclear cells with distinct basophilic granules.

CASE VI.—Thomas McK., aged ten years, had been in bad health for several months, with some cough. When admitted he was evidently in the first week of typhoid fever. There was

rather more bronchitis than usual, and the mucopurulent expectoration was occasionally blood stained. The blood count showed: 5,120,000 red blood corpuscles; 9,266 white blood corpuscles; and 75 per cent. of hemoglobin. The differential count: 69.4 per cent. polymorphonuclears; 10.6 per cent. mononuclears; 20 per cent. lymphocytes.

The microscopic examination: Canon stain—red corpuscles uniformly somewhat purplish in color. The leucocytes were large and small and the former were rather difficult to distinguish from the mononuclear cells. In the latter, the nucleus was deeply stained and the protoplasm light colored. In one case, however, a very large mononuclear contained a pale nucleus and deeply stained protoplasm. In the specimens fixed with bichlorid, one polymorphonuclear cell was found with distinct basophilic granules. A few cells were found which are recorded as "doubtful myelocytes."

CASE VII.—Laura G. had typhoid fever and developed a bronchopneumonia. The blood count was made late in the case when the convalescence was practically established. There were 5,200,000 red blood corpuscles; 20,928 white blood corpuscles; and 76 per cent. of hemoglobin. The differential count showed: 17.3 per cent. polymorphonuclears; 50.7 per cent. mononuclears; 32 per cent. lymphocytes.

The microscopic examination showed marked irregularity in shape and some excessively large red blood corpuscles; a few shadow cells and all of the red corpuscles poorly stained.

CASE VIII.—Harry B., aged about six years, was admitted in the second week of typhoid fever. There was slight bronchitis. The blood count was as follows: 4,200,000 red blood corpuscles; 7,000 white blood corpuscles; 78 per cent. hemoglobin. The differential count: 53.1 per cent. polymorphonuclears; 16.7 per cent. mononuclears; 30.2 per cent. lymphocytes. A second count was made during the convalescence from the typhoid fever and the following figures were obtained: 4,360,000 red blood corpuscles; 8,342 white blood corpuscles; 68 per cent. hemoglobin. The differential count: 52.9 polymorphonuclears; 16.4 per cent. mononuclears; 30.7 per cent. lymphocytes.

The microscopic examination of the stained specimens showed: red corpuscles large, irregular in shape and in size,

and one distinct nucleated corpuscle of a rather large size with central deeply staining nucleus, surrounded by a clear space. Among the leucocytes were several large forms with excentric nuclei having irregular outlines; they resembled myelocytes. A number of fragmented leucocytes were seen. In the specimen stained with Canon stain some polychromatophilia was seen. The microscopic examination of the specimen during convalescence showed nothing abnormal.

CASE IX.—Benjamin S., aged eight years, was admitted with well developed typhoid fever, and had a few râles indicative of bronchitis. There was paroxysmal cough which developed into distinct pertussis and an eruption of varicella occurred a week after admission. Examination of the blood at the time of admission showed: 3,808,000 red blood corpuscles; 20,800 white blood corpuscles; and 83 per cent. hemoglobin. The differential count: 81.6 per cent. polymorphonuclears, 6.2 per cent. mononuclears, 10.4 per cent. lymphocytes, and 1.8 per cent. eosinophiles.

The microscopic examination showed: red corpuscles normal in appearance for the most part but one distinct megaloblast, a number of microblasts, and some shadow corpuscles and fragmented cells were found.

In these cases the absence of leukocytosis, noted in adults, was found in all excepting three; and in these complications (severe bronchitis, pneumonia, and pertussis and varicella) were sufficient to explain the increased number of leucocytes. The differential counts of leucocytes were not characteristic but the occurrence of myelocytes in at least three of the cases is notable.

PERTUSSIS.

CASE I.—Marie G., aged twenty-two months, a rachitic child, was admitted with a cough, which had existed for six weeks. Sibilant râles were discovered in both lungs and characteristic whoops developed in a few days. The blood count showed 5,700,000 red blood corpuscles; 12,145 white blood corpuscles: 82 per cent. hemoglobin. The differential count: 40.8 per cent. polymorphonuclears; 27.8 per cent. mononuclears; 24 per cent. lymphocytes; 5.6 per cent. eosinophiles; 1.8 per cent. myelocytes.

Microscopic examination showed: the red corpuscles were equal in size and well-stained. There was much variability in

the appearance of the individual types of leucocytes; some of the mononuclear being very similar in appearance to lymphocytes; others more typical according to the ordinary description. Similarly, the lymphocytes varied from small bodies in which the nucleus and protoplasm were scarcely distinguishable to large forms approaching the large mononuclear. Both neutrophilic and eosinophilic myelocytes were observed.

CASE II.—X. Y., aged about four years, was admitted with whooping-cough. The blood count showed: 4,545,000 red blood corpuscles; 34,667 white blood corpuscles; 88 per cent. hemoglobin. The differential count: 29.2 per cent. polomorphonuclears; 17.4 per cent. mononuclears; 52.6 per cent. lymphocytes; 0.8 per cent. eosinophiles; 0.1 per cent. myelocytes.

Nothing of consequence beyond the existence of myelocytes in the microscopical examination.

CASE III.—Theodore W., aged six was first admitted to the hospital with malaria, but subsequently developed pertussis. At that time the blood count showed: 4,187,500 red blood corpuscles; 16,218 white blood corpuscles; 73 per cent. hemoglobin. The differential count: 41.4 per cent. polymorphonuclears; 19.5 per cent. mononuclears; 36.9 per cent. lymphocytes; 2.2 per cent. eosinophiles. The microscopic examination: normal red cells; a number of degenerated mononuclear cells; otherwise no abnormality.

In three cases the most striking peculiarity was the marked increase of lymphocytes. This may be of interest in connection with the supposed disease of the lymph glands in this disease.

VARICELLA.

CASE I.—William G., aged seven and a half years, developed varicella in the house. The child was of rather strumous appearance. During the attack, the blood count showed: 4,743,700 red blood corpuscles; 7,466 white blood corpuscles, and 75 per cent. hemoglobin. The differential count: 56.5 per cent. polymorphonuclears; 19.1 per cent. mononuclears; 23.4 per cent. lymphocytes; 1 per cent. eosinophiles.

The microscopic examination showed some inequality of red corpuscles with occasional macrocytes and slight polychromatophilia.

CASE II.—Harry B., aged two years and eight months, was first admitted to the hospital with typhoid fever from which convalescence was rapid. He developed varicella two weeks and five days after admission, and had a copious eruption. During the attack, the blood count showed: 7,440 leucocytes. The count of red corpuscles and the amount of hemoglobin were not preserved. The differential count showed: 46 per cent. polymorphonuclears; 16.4 per cent. mononuclears; 36.4 per cent. lymphocytes; 1 per cent. eosinophiles; and 0.2 per cent. myelocytes. There was nothing of any consequence in the microscopical examination.

CASE III.—William M., aged six months, ill developed child, with some cough, and bronchial râles, developed varicella in the hospital, and afterwards had pneumonia and died. During the period of varicella, the blood count showed: 5,300,000 red blood corpuscles; 19,360 white blood corpuscles; and 98 per cent. hemoglobin. The differential count showed: 70.2 per cent. polymorphonuclears; 16.6 per cent. mononuclears; 12.8 per cent. lymphocytes; 0.4 per cent. eosinophiles.

The microscopic examination showed well stained and normal red corpuscles; one nucleated red corpuscle was found. The mononuclear leucocytes were frequently degenerated or fragmented in appearance; some were distinctly so and had jagged outline. There was occasional hyperchromatosis.

CASE IV.—William S., aged four years, developed varicella in the hospital. The blood count showed: 5,330,000 red blood corpuscles; 12,800 white blood corpuscles; and 90 per cent. hemoglobin. The differential count showed: 44.9 per cent. polymorphonuclears; 20.5 per cent. mononuclears; 33.8 per cent. lymphocytes; 0.8 per cent. eosinophiles.

The microscopic examination: red blood corpuscles stained poorly, and were somewhat irregular in shape. The mononuclear cells were very variable in size and many were excessively large. Some of these large forms contained granular basophilic protoplasm; others were entirely clear. The same characters of protoplasm were observed in the lymphocytes, and attached to the latter were occasionally granular particles entirely outside of the cells but attached by small threads.

TUBERCULOUS CARIES WITH COLD ABSCESS.

Edward F., aged about ten years, was admitted to the surgical ward with a mass in the abdomen which was regarded as a sold growth, but which subsequently showed itself to be fluctuating, and eventually was found to be a tuberculous collection secondary to necrosis of the lumbar vertebræ.

The blood count showed: red blood corpuscles 4,500,000; white blood corpuscles 20,579; hemoglobin 66 per cent. The differential count showed: polymorphonuclear cells 70.3 per cent.; mononuclear 18.1 per cent.; lymphocytes 10.7 per cent.; eosinophiles 0.9 per cent.

The microscopic examination of the stained specimens showed some distortion of the red corpuscles. Several polymorphonuclear cells with distinct basophilic granules were discovered. These were quite distinctly different from the basophilic granules of the mononuclear cells, which were fine and indistinctly stained. The ones in the polymorphonuclear on the contrary were coarse and occasionally larger than the granules of the eosinophiles. The mononuclear cells generally showed a basophilic protoplasm with occasionally fine but indistinct granulation. The differentiation of the mononuclear cells and lymphocytes was very difficult, and the differential count is possibly erroneous in giving too great a proportion of mononuclear cells. There was no polychromatophilia.

ACUTE RHEUMATISM.

Jacob F., aged five years, was admitted with pain in the back and legs. The legs were exceedingly tender to the touch. The ankles were slightly swollen and tender; there was constipation. The history indicated a subsiding articular rheumatism.

The blood count showed: 4,355,000 red blood corpuscles; 7,022 white blood corpuscles, and 75 per cent. hemoglobin. The differential count showed: polymorphonuclears 59 per cent., mononuclears 22.4 per cent., lymphocytes 17.6 per cent., eosinophiles 1 per cent.

On microscopic examination of the stained specimens the red corpuscles were found deeply colored but entirely normal in appearance. Among the polymorphonuclear leucocytes, several were found with deeply staining protoplasm (somewhat acidophilic) and with vacuoles. Vacuolated mononuclear cells were

also seen. One of the latter forms contained numerous vacuoles and another mononuclear cell contained very distinct and rather coarse basophilic granules scattered throughout the cell and over the nucleus.

NOMA.

CASE I.—Helen O'D., aged about seven years, was admitted with a history of vague illness beginning about two weeks before entrance into hospital. There had been slight cough and abdominal pain. The temperature on admission was 103.2° F. The spleen was slightly enlarged, and there were a few suspicious spots on the abdomen. Tongue coated but not characteristic; lungs clear. The appearance was like that of a typhoid case, but the history was uncertain and Widal test negative. Delirium occurred and the face became swollen. A bad tooth was discovered and noma developed. Curetted and cauterized. Rapid progress, extreme gangrene and death after eleven days.

The blood counts showed:

1st count on admission: Red blood corpuscles, 5,380,000; leucocytes, 9,822; hemoglobin, 80 per cent.

2d count three days later: Red blood corpuscles, 4,185,000; leucocytes, 5,058; hemoglobin, 65 per cent.

3d count day before death: Red blood corpuscles, 3,260,000; leucocytes, 12,144; hemoglobin, 58 per cent.

The differential counts were as follows:

1st count: Polymorphonuclears, 86.4 per cent.; mononuclears, 7.4 per cent.; lymphocytes, 5.8 per cent.; eosinophiles, 0.4 per cent.

2d count: Polymorphonuclears, 72.5 per cent.; mononuclears, 13 per cent.; lymphocytes, 14.5 per cent.

3d count: Polymorphonuclears, 74.5 per cent.; mononuclears, 16.3 per cent.; lymphocytes, 9.2 per cent.

The microscopic examination at the time of the first count showed some poikilocytosis and dark chromatin masses in the polymorphonuclear leucocytes. At the second count nothing of note was observed excepting a fine chromatin net work in the protoplasm of many polymorphonuclear cells, suggesting basophilic granules; and a similar condition in lymphocytes as well as projecting strands with distinctly bulbous extremities;

occasional hyperchromatosis was found in the mononuclears. The neutrophilic granules varied in coarseness in different polymorphonuclear cells.

Third examination: Irregularity of the red corpuscles in size and shape; uneven staining. Occasional pseudovacuolation and slight polychromatophilia; megalocytes. All forms of the leucocytes when stained with Canon's stain showed here and there basophilic granules, which in some of the mononuclears seemed quite clearly to be nodal points in a protoplasmic network. The polymorphonuclear cells frequently contained basophilic granules and some had very pronounced granulations of this sort.

BRONCHITIS.

CASE I.—Louise B., aged four years, was admitted to the hospital with fever and the evidences of pulmonary disease. There were scattered moist râles on both sides and a suspicion of dulness at the right base, but no positive dulness. The diagnosis of catarrhal pneumonia was made. Examination of the blood showed 5,126,000 red blood corpuscles; 14,619 white blood corpuscles; 92 per cent. hemoglobin.

CASE II.—Jennie H., aged five years, had suffered with purulent otitis since her first year. Her mother had died of phthisis, and there had been five or six miscarriages. Five or six children died young. The diagnosis of bronchitis and subacute pneumonia was made. The blood examination showed 3,875,000 red blood corpuscles; 15,300 white blood corpuscles, and 65 per cent. hemoglobin. The differential count: 63.4 per cent. polymorphonuclears; 12.5 per cent. mononuclears; 22.9 per cent. lymphocytes, and 1.2 per cent. eosinophiles.

The microscopical study of the specimens: Canon's stain—red corpuscles normal; mononuclear leucocytes very large with poorly stained nucleus, and occasionally deep granular protoplasm; lymphocytes both large and small; protoplasm stained deeply with methylene blue. Nothing of interest observed in the specimens fixed and stained by other methods.

CASE III.—Mary McC., aged four years, was admitted with acute bronchitis and slight diarrhea. There were râles on both sides of the chest; also some slight acute tonsillitis and pharyngitis, with enlargement of the lymphatic glands of the neck. The blood count showed 5,390,000 red blood corpuscles; 19,226

white blood corpuscles, and 96 per cent. hemoglobin. The differential count: 74.7 per cent. polymorphonuclears; 11.2 per cent. mononuclears; 12.9 per cent. lymphocytes; 1.2 per cent. eosinophiles.

The stained specimens: Canon's stain—red corpuscles about normal; white corpuscles showed nothing striking, excepting the pallor of the nuclei of the mononuclear forms and a tendency to granular basophilic protoplasm in the same cells. Nothing of consequence in the specimens prepared by other methods.

CASE IV.—Louis S., aged three years, was admitted with acute bronchitis. The blood count showed 5,010,000 red blood corpuscles; 12,909 white blood corpuscles; 63 per cent. hemoglobin. The differential count: 69.4 per cent. polymorphonuclears; 12.6 per cent. mononuclears; 18 per cent. lymphocytes.

The examination of the stained specimens: Canon stain—red corpuscles normal in appearance; mononuclear leucocytes frequently presented an unusually pale nucleus and granular protoplasm.

CASE V.—Theodore W., aged six years, was admitted with acute bronchitis. The blood count showed 4.958,000 red blood corpuscles; 12,691 white blood corpuscles; 83 per cent. hemoglobin. The differential count: 61.3 per cent. polymorphonuclears; 6.9 per cent. mononuclears; 29.9 per cent. lymphocytes; 1.9 per cent. eosinophiles.

The examination of the stained specimens: Canon stain—red corpuscles normal; lymphocytes occasionally had basophilic granules, but more often the protoplasm was profusely basophilic.

CASE VI.—Veronica D., aged three years, was admitted with subacute bronchitis. She had had pneumonia, but the impairment of the lung and the active signs of pneumonia had entirely disappeared. There was enlargement of both tonsils. The blood count showed 3,775,000 red blood corpuscles; 14,507 white blood corpuscles; 82 per cent. hemoglobin. The differential count: 87 per cent. polymorphonuclears; 9.4 per cent. mononuclears; 3.6 per cent. lymphocytes.

The microscopical examination: the red corpuscles were normal; white corpuscles—there were two distinct varieties of lymphocytes, large and the small; the protoplasm of the former

being without granules, that of the latter stained a bluish color. The larger forms were difficult to distinguish from mononuclear cells.

CASE VII.—Bessie B., aged seven and one-half years was admitted to the hospital with acute bronchitis and had moderate continuous fever. The child also had seat worms. The blood count showed 3,880,000 red blood corpuscles; 12,835 leucocytes and 78 per cent. of hemoglobin. The differential count showed: 52.3 per cent. polymorphonuclears; 15.1 per cent. mononuclears; 25.3 lymphocytes; 7.3 per cent. eosinophiles. The microscopic examination showed: some irregularity in the staining, and size and shape of the red corpuscles, with occasional polychromatophilia. The white corpuscles showed no peculiarities.

The moderate leukocytosis is the only notable condition discovered. In two or three of the cases it is likely that there were patches of bronchopneumonia, though none could be classed as an instance of pneumonia in a strict sense.

PLEURAL EFFUSION.

CASE I.—Harry B., aged twenty-three months, was admitted with pleural effusion. There was marked dyspnea and slight cyanosis, and the right pleural cavity was filled to the second rib. The legs were moderately edematous; the hands less so. The blood count showed: 3,755,000 red blood corpuscles; 13,610 white blood corpuscles; 70 per cent. hemoglobin. The differential count: 37.2 per cent. polymorphonuclears; 34.5 per cent. mononuclears; 27.1 per cent. lymphocytes; 1.2 per cent. eosinophiles.

Microscopically, the red corpuscles were found unequal in size. Some distinct megaloblasts and microcytes were observed; a few polychromatophilic corpuscles were seen. The leucocytes showed no abnormalities.

ENTERITIS.

CASE I.—Eva P., aged ten months, was admitted with marked enteritis. The stools were filled with mucus, and were occasionally blood-streaked. There was a slight cough, and also a slight vaginitis. The blood count showed: 4,060,000 red blood corpuscles; 27,666 white blood corpuscles; and 65 per cent. hemoglobin. The differential count showed: 29 per cent.

polymorphonuclears, [illegible] per cent. mononu[illegible] cent lymphocytes, [illegible] per cent. eosinophiles.

The microscopical examination: Canon st[illegible] corpuscles slightly irregular in size; some polych[illegible] Lymphocytes contained very dark nuclei; their pro[illegible] usually granular and generally basophilic. Son[illegible] nuclei presented themselves in ring forms.

[illegible] aged six years was an ill [illegible] child with a bad family history. The child was ver[illegible] and there were enlarged glands in the axilla, groins and [illegible] region. The appearance was that of profound inanitio[illegible] blood [illegible] red blood corpuscles; [illegible] per cent. hemoglobin. [illegible] per cent. polymorphonuclears; [illegible] per cent. lymphocytes.

The [illegible] red corpuscles nor[illegible] white blood [illegible] large mononuclear [illegible] and a distincti[illegible] between [illegible] could not be made. Th[illegible] [illegible] cells. The lymphocyte[illegible] [illegible] The specimen stained [illegible] white cells.

[illegible] was admitted as a [illegible] tubercular history, [illegible] milk. Sometime [illegible] developed varicella [illegible] chronic enteritis [illegible] to the varicella [illegible] white blood [illegible] differential count [illegible] per cent. [illegible] abnormality of [illegible] occasionally [illegible] years was [illegible] due to uxuris. [illegible]

MITRAL HEART DISEASES.

CASE I.—Emily B., aged eleven and a half years, was admitted to the hospital with double mitral valvular disease. There were occasional attacks of cyanosis; no edema nor other signs of failing compensation. The blood count showed: red corpuscles 4,390,000; leucocytes 13,658; hemoglobin 79 per cent. The differential count showed: polymorphonuclears 68.6 per cent.; mononuclears 13 per cent.; lymphocytes 18.2 per cent.; eosinophiles 0.2 per cent.

The microscopic examination showed as follows: picric acid, eosin, hematoxylon specimen: protoplasm of mononuclears and lymphocytes stained dark blue; some vacuolation; lymphocytes large and small; red corpuscles normal. Canon stain of heat—fixed specimen: protoplasm and nucleus of mononuclears hard to distinguish; slight basic granulation of the protoplasm; lymphocytes of two sizes, the larger being the paler nucleus; protoplasm in both forms dark blue.

CASE II.—Thomas S., aged six years, had rheumatic valvular disease, double mitral. He was subject to attacks of dyspnea and anasarca. The blood count showed: red corpuscles 4,975,000; leucocytes 20,587; hemoglobin 70 per cent. The differential count: polymorphonuclears 58.4 per cent.; mononuclears 13.8 per cent.; lymphocytes 25.4 per cent.; eosinophiles 2.2 per cent.; myelocytes 0.2 per cent.

The microscopic examination showed as follows: Canon stain with heat fixation. The mononuclears were of two kinds, one with pale nucleus and granular protoplasm, the other with a dark nucleus and unstained protoplasm, a few contained distinct basophilic granulations; lymphocytes were variable in size: the larger showing a basophilic protoplasm; one distinct myelocyte was found.

RACHITIS.

CASE I.—Emma F., aged nineteen months, had marked signs of rickets. The blood count showed: red blood corpuscles 5,170,000; white blood corpuscles 11,911; 78 per cent. hemoglobin. The differential count showed: polymorphonuclears 42 per cent.; mononuclears 18.4 per cent.; lymphocytes 36.6 per cent.; eosinophiles 2.6 per cent.; myelocytes 0.4 per cent.

The microscopic examination of the stained preparations showed normal red corpuscles, but several distinct peculiarities in the leucocytes. The polymorphonuclears were variable in size, the larger forms having pale nuclei and the smaller ones nuclei of the ordinary appearance. Several contained distinct basophilic granulations which were deeply stained. The same form of granules was found in several mononuclear cells. The lymphocytes were present in two varieties, some being very small with a densely stained nucleus and little protoplasm, and others large and containing pale nuclei with basophilic protoplasm. The eosinophile cells were very large and unusually full of granules. The myelocytes were exceptionally large with oval nuclei placed to one side of the cell in fine granules.

CASE II.—William G., aged twenty months, was admitted with coryza, cough, and gastric disturbances. There was some diarrhea, and the child was greatly emaciated. It was reported that he had whooped, but no confirmation of this could be obtained. There were crackling râles in the chest. The child was decidedly rachitic.

Examination of the blood showed: red blood corpuscles 6,180,000 (?); leucocytes 29,557; 64 per cent. hemoglobin. The differential count showed: polymorphonuclears 44.7 per cent.; mononuclears 19.6 per cent.; lymphocytes 34.5 per cent.; eosinophiles 1.2 per cent.

The large proportion of lymphocytes and mononuclears in these cases was the most notable condition. Several other cases of the series examined were rachitic, but this condition was subordinate to some other disease and the cases have therefore been placed under other headings.

ECZEMA.

William L., aged two years and three months, had facial eczema which had lasted for three months. Later there were patches on the abdomen and other parts of the body.

The first blood count showed: red blood corpuscles 5,200,000; white blood corpuscles 22,000; 76 per cent. of hemoglobin. Two subsequent counts of the leucocytes showed 17,541 and 10,947. The differential counts at these three examinations showed: No. 1.—37.1 per cent. polymorphonuclears; 21 per cent. mononuclears; 35 per cent. lymphocytes; 7.9 per cent.

eosinophiles. No. 2.—59 per cent. polymorphonuclears; 11.6 per cent. mononuclears; 23.7 per cent. lymphocytes; 5.7 per cent. eosinophiles. No. 3.—61.2 per cent. polymorphonuclears; 17.0 per cent. mononuclears; 14.0 per cent. lymphocytes; 7.8 per cent. eosinophiles.

The microscopic examination showed some irregularity in the red blood corpuscles with distinct microcytes. The leucocytes stained well, while the protoplasm of the polymorphonuclears was pinkish in the eosin and methylene blue stains. The protoplasm of the lymphocytes was basophilic. Occasionally dark basophilic granules were found outside the lymphocytes and attached by narrow pedicles. These had the appearance of extrusion. The protoplasm of the mononuclear cells was faintly basophilic and occasionally distinct mast-cell granulations were found. The eosinophiles were prominent and of large size.

FOCAL EPILEPSY.

Harry B., aged eight years, was admitted with a history of convulsions beginning in the leg. There was no palsy nor atrophy. His station was good and reflexes normal.

Examination of the blood showed: 4,662,500 red blood corpuscles; 11,911 white blood corpuscles; and 85 per cent. hemoglobin.

The differential count showed: polymorphonuclears 48.5 per cent.; mononuclears 20 per cent.; lymphocytes 30.3 per cent.; eosinophiles 1.2 per cent.

CONVULSIONS.

A. D., aged two months, was admitted to the hospital with a history of having had convulsions. Nothing very definite was known regarding the nature of these. There was some looseness of the bowels, but not any distinct signs of disease.

The blood count taken 20 minutes after a convulsion showed: red blood corpuscles 2,520,000; leucocytes 8,800; hemoglobin 60 per cent. The differential count showed: polymorphonuclears 42.8 per cent.; mononuclears 23.2 per cent. lymphocytes 33.6 per cent.; eosinophiles 0.4 per cent.

The microscopic examination showed irregularity, degeneration, and polychromatophilia of the red corpuscles. Several of the polymorphonuclear leucocytes contained distinct basophilic granules.

SPASTIC CEREBRAL PALSY.

Victoria D., aged about five years, has been in the hospital for some time with symptoms of spastic cerebral paralysis. There were no convulsive seizures.

The blood examination showed: 4,276,250 red blood corpuscles; 15,808 white blood corpuscles; and 85 per cent. of hemoglobin.

The differential count showed: polymorphonuclears 40.6 per cent.; mononuclears 9.9 per cent.; lymphocytes 49.1 per cent.; eosinophiles 0.4 per cent.

The microscopic examination of the stained specimen showed well stained and slightly irregular red corpuscles. White corpuscles normal in every respect.

CHRONIC MENINGITIS.

Margaret M., aged three and a half years, had doubtful symptoms of chronic meningitis with occasional convulsions. She had taken potassium iodid and presented indications of iodism.

The blood count: red corpuscles 5,412,500; leucocytes 21,333; hemoglobin 85 per cent.; specific gravity 1068.

The differential count: polymorphonuclears 66 per cent. mononuclears 10.6 per cent.; lymphocytes 23.2 per cent.; eosinophiles 0.2 per cent.

The microscopic examination showed some irregularity in the shape and size of the red cells. The protoplasm of the mononuclear cells was clear, that of the lymphocytes stained deeply with methylene blue.

SUMMARY.

Case Number.	Hemoglobin.	Erythrocytes.	Leucocytes.	Polymorphonuclear.	Mononuclear.	Lymphocytes.	Eosinophiles.	Myelocytes.	Diagnosis and Remarks.
1	85 per cent.	4,460,000	29,200	87.0 per cent.	9.4 per cent.	3.6 per cent.	———	0.4 per cent.	Helen D. Croupous pneumonia.
2*a*	56 per cent.	4,332,000	68,000	59.6 per cent.	6.4 per cent.	30.4 per cent.	1.4 per cent.	2.2 per cent.	Lazer T. Croupous pneumonia and rachitis.
2*b*	55 per cent.	———	87,200	49.6 per cent.	8.8 per cent.	39.6 per cent.	1.4 per cent.	0.6 per cent.	Myelocytes, 0.5. Eosinophilic myelocytes, 0.1.
2*c*	46 per cent.	3,440,000	27,824	47.5 per cent.	13.9 per cent.	37.4 per cent.	0.8 per cent.	0.4 per cent.	
3*a*	85 per cent.	4,540,000	35,200	71.6 per cent.	10.5 per cent.	17.9 per cent.	———	———	Sarah M. Croupous pneumonia.
3*b*	90 per cent.	4,312,500	18,606	70.5 per cent.	13.3 per cent.	16.2 per cent.	———	0.2 per cent.	10 days later.
4	78 per cent.	5,025,000	34,688	86.1 per cent.	6.2 per cent.	7.6 per cent.	———	0.1 per cent.	William M. Croupous pneumonia (double).
5	70 per cent.	4,962,500	32,160	84.3 per cent.	8.1 per cent.	7.6 per cent.	———	———	Annie S. Croupous pneumonia, meningitis (?).
6	69 per cent.	3,386,000	20,400	52.6 per cent.	15.9 per cent.	31.5 per cent.	———	———	Josephine G. Croupous pneumonia and axillary abscess.
7	83 per cent.	3,506,200	50,917	73.0 per cent.	11.3 per cent.	14.7 per cent.	0.9 per cent.	0.1 per cent.	Jacob H. Croupous pneumonia.
1	77 per cent.	4,125,500	27,636	78.0 per cent.	13.5 per cent.	8.5 per cent.	———	———	Selina P. Typhoid fever.
2*a*	77 per cent.	5,025,000	6,966	85.9 per cent.	8.1 per cent.	6.0 per cent.	———	0.2 per cent.	Theresa K. Typhoid fever (before tub).
2*b*	———	———	13,066	———	———	———	———	———	Theresa K. Typhoid fever (after tub) increase in the polymorphonuclear cells.

Case Number.	Hemoglobin.	Erythrocytes.	Leucocytes.	Polymorphonuclear.	Mononuclear.	Lymphocytes.	Eosinophiles.	Myelocytes.	Diagnosis and Remarks.
3	78 per cent.	3,320,000	6,948	55.3 per cent.	9.4 per cent.	34.3 per cent.	0.9 per cent.	———	Rachel H. Typhoid fever. Second relapse.
4*a*	70 per cent.	4,565,000	4,207	75.4 per cent.	11.6 per cent.	12.4 per cent.	———	0.6 per cent.	Ambrose L. Typhoid fever, immediately before tub bath.
4*b*	———	———	3,800	78.7 per cent.	9.5 per cent.	10.2 per cent.	0.8 per cent.	0.8 per cent.	Ambrose L. Typhoid fever, two hours later.
4*c*	80 per cent.	4,830,000	12,320	66.5 per cent.	11.7 per cent.	21.6 per cent.	0.2 per cent.	———	Ambrose L. Typhoid fever, in convalescence.
5*a*	70 per cent.	3,716,000	6,880	36.3 per cent.	27.3 per cent.	35.8 per cent.	0.6 per cent.	———	Bessie J. Typhoid fever.
5*b*	70 per cent.	3,850,000	9,840	———	———	———	———	———	" " relapse and after the tub bath.
6	75 per cent.	5,120,000	9,266	69.4 per cent.	10.6 per cent.	20.0 per cent.	———	———	Thomas McK. Typhoid fever.
7	76 per cent.	5,200,000	20,928	17.3 per cent.	50.7 per cent.	32.0 per cent.	———	———	Laura G. Typhoid fever and bronchopneumonia.
8*a*	78 per cent.	4,200,000	7,000	53.1 per cent.	16.7 per cent.	30.2 per cent.	———	———	Harry B. Typhoid fever.
8*b*	68 per cent.	4,360,000	8,342	52.9 per cent.	16.4 per cent.	30.7 per cent.	———	———	" " " during convalescence.
9	83 per cent.	3,808,000	20,800	81.6 per cent.	6.2 per cent.	10.4 per cent.	1.8 per cent.	———	Benjamin S. Typhoid fever and bronchitis.
1	82 per cent.	5,700,000	12,145	40.8 per cent.	27.8 per cent.	24.0 per cent.	5.6 per cent.	1.8 per cent.	Marie G. Pertussis and rachitis.
2	88 per cent.	4,545,000	34,666	29.2 per cent.	17.4 per cent.	52.6 per cent.	0.8 per cent.	0.1 per cent.	X. Y. Pertussis.

Case Number.	Hemoglobin.	Erythrocytes.	Leucocytes.	Polymorphonuclear.	Mononuclear.	Lymphocytes.	Eosinophiles.	Myelocytes.	Diagnosis and Remarks.
3	73 per cent.	4,187,500	16,218	41.4 per cent.	19.5 per cent.	36.9 per cent.	2.2 per cent.	———	Theodore W. Pertussis and malaria, convalescence.
1	75 per cent.	4,743,700	7,466	56.5 per cent.	19.1 per cent.	23.4 per cent.	1.0 per cent.	———	William G. Varicella.
2	———	———	7,440	46.0 per cent.	16.4 per cent.	36.4 per cent.	1.0 per cent.	0.2 per cent.	Harry B. Varicella.
3	98 per cent.	5,300,000	19,360	70.2 per cent.	16.6 per cent.	12.8 per cent.	0.4 per cent.	———	William M. Varicella, pneumonia.
4	90 per cent.	5,330,000	12,800	44.9 per cent.	20.5 per cent.	33.8 per cent.	0.8 per cent.	———	William S. Varicella.
1	66 per cent.	4,500,000	20,579	70.3 per cent.	18.1 per cent.	10.7 per cent.	0.9 per cent.	———	Edward F. Tubercular caries with cold abscess.
1	75 per cent.	4,355,000	7,022	59.0 per cent.	22.4 per cent.	17.6 per cent.	1.0 per cent.	———	Jacob F. Acute rheumatism (subsiding).
1*a*	80 per cent.	5,380,000	9,822	86.4 per cent.	7.4 per cent.	5.8 per cent.	0.4 per cent.	———	Helen O'D. Noma, on admission.
1*b*	65 per cent.	4,185,000	5,058	72.5 per cent.	13.0 per cent.	14.5 per cent.	———	———	" " three days later
1*c*	58 per cent.	3,260,000	12,144	74.5 per cent.	16.3 per cent.	9.2 per cent.	———	———	" " the day before death.

Case Number.	Hemoglobin.	Erythrocytes.	Leucocytes.	Polymorphonuclear.	Mononuclear.	Lymphocytes.	Eosinophiles.	Myelocytes.	Diagnosis and Remarks.
1	92 per cent.	5,126,000	14,619	———	———	———	———	———	Louisa B. Bronchitis, catarrhal pneumonia.
2	65 per cent.	3,875,000	15,300	63.4 per cent.	12.5 per cent.	22.9 per cent.	1.2 per cent.	———	Jennie H. Bronchitis, subacute pneumonia.
3	96 per cent.	5,390,000	19,226	74.7 per cent.	11.2 per cent.	12.9 per cent.	1.2 per cent.	———	Mary McC. Bronchitis (acute), slight tonsillitis and pharyngitis.
4	93 per cent.	5,010,000	12,909	69.4 per cent.	12.6 per cent.	18.0 per cent.	———	———	Louis S. Bronchitis (acute).
5	83 per cent.	4,958,000	12,690	61.3 per cent.	6.9 per cent.	29.9 per cent.	1.9 per cent.	———	Theodore W. Bronchitis (acute).
6	82 per cent.	3,775,000	14,507	87.0 per cent.	9.4 per cent.	3.6 per cent.	———	———	Veronica D. Bronchitis (subacute).
7	78 per cent.	3,880,000	12,835	52.3 per cent.	15.1 per cent.	25.3 per cent.	7.3 per cent.	———	Bessie B. Bronchitis (acute).
1	70 per cent.	3,755,000	13,610	37.2 per cent.	34.5 per cent.	27.1 per cent.	1.2 per cent.	———	Harry B. Pleural effusion.
1	65 per cent.	4,060,000	27,666	29.0 per cent.	38.3 per cent.	31.8 per cent.	0.9 per cent.	———	Eva P. Chronic catarrh, enteritis and vaginitis.
2	68 per cent.	5,050,000	16,081	76.0 per cent.	10.8 per cent.	13.2 per cent.	———	———	Jennie M. Chronic enteritis and inanition.
3	97 per cent.	4,640,000	26,800	59.6 per cent.	15.3 per cent.	24.9 per cent.	0.2 per cent.	———	Edith W. Chronic enteritis and inanition.
4	94 per cent.	5,125,000	9,499	———	———	———	———	———	William G. Enteritis following intestinal parasites.

Case Number.	Hemoglobin	Erythrocytes.	Leucocytes.	Polymorphonuclear.	Mononuclear.	Lymphocytes.	Eosinophiles.	Myelocytes	Diagnosis and Remarks
1	79 per cent.	4,190,000	13,658	68.6 per cent.	13.0 per cent.	18.2 per cent.	0.2 per cent.	———	Emily B. Mitral stenosis and regurgitation.
2	70 per cent.	4,975,000	20,587	58.4 per cent.	13.8 per cent.	25.4 per cent.	2.2 per cent.	0.2 per cent.	Thomas S. Mitral stenosis and regurgitation.
1	78 per cent.	5,170,000	11,911	42.0 per cent.	18.4 per cent.	36.6 per cent.	2.6 per cent.	0.4 per cent	Emma F. Rachitis.
2	64 per cent.	6,180,000	29,557	44.7 per cent.	19.6 per cent.	34.5 per cent.	1.2 per cent.	———	William G. " severe diarrhea, emaciation.
1*a*	76 per cent.	5,200,000	22,000	37.1 per cent.	21.0 per cent.	35.0 per cent.	7.9 per cent.	———	William L. Eczema.
1*b*	———	———	17,541	59.0 per cent.	11.6 per cent.	23.7 per cent.	5.7 per cent.	———	" "
1*c*	———	———	10,947	61.2 per cent.	17.0 per cent.	14.0 per cent.	7.8 per cent.	———	" "
1	85 per cent.	4,662,500	11,911	48.5 per cent.	20 0 per cent.	30.3 per cent.	1.2 per cent.	———	Harry B. Focal epilepsy.
1	60 per cent.	2,520,000	8,800	42.8 per cent	23.2 per cent.	33.6 per cent.	0.4 per cent.	———	Antonio D. Convulsions.
1	85 per cent.	4,276,250	15,808	40.6 per cent.	9.9 per cent.	49.1 per cent.	0.4 per cent.	———	Victoria D. Spastic cerebral palsy.
1	85 per cent.	5,412,500	21,333	66.0 per cent.	10.6 per cent.	23.2 per cent.	0.2 per cent.	———	Margaret M. Chronic meningitis.

41
25